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SURGICAL DISEASES OF THE SPINAL CORD, MEMBRANES, AND NERVE ROOTS

Symptoms, Diagnosis, and Treatment

SURGICAL DISEASES OF THE SPINAL CORD, MEMBRANES, AND NERVE ROOTS

SYMPTOMS, DIAGNOSIS, AND TREATMENT

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PREFACE

DURING THE PAST THREE DECADES, THE KNOWLEDGE OF THE SYMPTOMATOLOGY OF DISEASES AND INJURIES OF THE SPINAL CORD has been much advanced, and the field of the surgery of the spinal cord and nerve roots has been greatly extended. The symptoms produced by localized disease or by pathological processes in the neighborhood of the spinal cord are now better understood, and the indications for therapy in a variety of disease processes which cause pain, spasticity or other motor disturbances have become more exact. In some diseases of the spinal cord there is, as yet, no known method of treatment, but in an increasing number of patients relief can be obtained through surgical measures. There is, therefore, justification for a monograph on what may be called the surgical diseases of the spinal cord.

This volume contains much that was not known when I wrote "Diseases of the Spinal Cord" (1916) and "Tumors of the Spinal Cord" (1925). It may, therefore, be considered a new and revised edition of the previous books. In this volume it is my object to state concisely the essential features of diseases and injuries of the spinal cord, membranes, and nerve roots, which the surgeon may encounter, as well as to give the indications for operative intervention and to describe the technique of operations upon the spinal cord and nerve roots.

The facts that are presented are based upon a not inconsiderable personal experience, and upon a careful study of the literature of the subject. In addition to giving full credit for the work of foreign observers, I have endeavored to utilize as fully as possible the publications of American neurologists, neurosurgeons, and roentgenologists to whose industry and investigations many of the advances in this field of endeavor have been due.

The majority of the patients whose records have been used for the statements made were seen and studied on the neurological and neurosurgical services of the Neurological Institute of New York, during a period of more than thirty years. This volume could not be published without an expression of appreciation to the neurologists of the Neurological Institute—the late Drs. Charles L. Dana, Pearce Bailey, Frederick Peterson and Frederick Tilney, and Drs. Bernard Sachs, Joseph Collins, Foster Kennedy, Walter Timme, Edwin G. Zabriskie, and many others too numerous to mention.

As the progress in roentgenographic diagnosis now occupies an important place in a discussion of the diagnosis of lesions of the spinal cord, a special chapter by Dr. Cornelius G. Dyke has been made a part of this book. As the histological classification of tumors of the spinal cord and membranes has been changed almost entirely since the monograph on "Tumors of the Spinal Cord" was published about fifteen years ago, a special chapter on the pathology of cord tumors has been written by Dr. Abner Wolf. The chapters by Dr. Dyke and by Dr. Wolf are profusely illustrated so as to serve as monographs on and atlases of the roentgenographic changes in the spine and of the pathological nature of spinal cord tumors.

I am glad to express my thanks to my secretary, Miss Edna Brucks, for great help in the preparation of the manuscript, and to Mr. Alfred Feinberg who made a large number of artistic illustrations for the book.

CHARLES A. ELSBERG

New York, N.Y.

CONTENTS

PREFACE v

I. I. Surgical Anatomy, Normal and Pathological Physiology of the Spinal Cord 1

II. Diagnosis of Localized Spinal Diseases in Which Surgical Procedures May Be Necessary 18

II. Lumbar Puncture and the Effects of the Withdrawal of the Cerebrospinal Fluid 25

III. The Roentgen Ray Diagnosis of Diseases of the Spinal Cord, Meninges, and Vertebrae . . . . *By Cornelius G. Dyke* 42

IV. Injuries of the Spinal Cord and Nerve Roots 123

V. Congenital Abnormalities of the Spine and Spinal Cord 163

VI. Inflammatory Diseases 185

VII. Tumors of the Spinal Cord, Nerve Roots, and Membranes. I. Frequency and Incidence 208

VIII. Tumors of the Spinal Cord, Nerve Roots, and Membranes. II. Pathology . . . . *By Abner Wolf* 231

IX. Tumors of the Spinal Cord, Nerve Roots, and Membranes. III. Symptomatology 365

X. Tumors of the Spinal Cord, Nerve Roots, and Membranes. IV. Diagnosis 405

XI. Tumors of the Spinal Cord, Nerve Roots, and Membranes. V. Treatment 439

XII. Tumors of the Spinal Cord, Nerve Roots, and Membranes. VI. Cysts: Herniation of Nucleus Pulposus: Gumma 461

XIII. Primary and Secondary Diseases of the Vertebrae (Including Vertebral Tumors) Which May Produce Spinal Cord Disturbances 482

XIV. Primary Abnormalities of the Spinal Blood Vessels 503

XV. Laminectomy 517
XVI. Nonoperative and Operative Procedures for the Relief of Intractable Pain 535
XVII. Operative Procedures for the Relief of Spastic Conditions and Other Motor Disturbances 544
Appendix 555
References 559
Index of Authors 585
Index of Subjects 589

SURGICAL DISEASES OF THE SPINAL CORD, MEMBRANES, AND NERVE ROOTS

Symptoms, Diagnosis, and Treatment

CHAPTER I

I. SURGICAL ANATOMY, NORMAL AND PATHOLOGICAL PHYSIOLOGY OF THE SPINAL CORD

FOR A DETAILED ACCOUNT OF THE ANATOMY OF THE SPINE AND SPINAL CORD, THE READER IS REFERRED TO TEXTBOOKS OF ANATOMY. In this and the next section, references will be made to a few facts of practical importance.

The Vertebral Column. In the cervical and upper dorsal regions, the vertebrae are smaller than in other parts of the spinal column. The spinous processes of the cervical vertebrae are bifid at their tips, and the vertebrae are rather freely movable, the one upon the other. This makes the removal of spinous processes and laminae most simple in the cervical region. The thoracic vertebrae are more fixed upon each other, and the spinous processes and laminae overlap, so that the removal of spines and laminae is most easily accomplished in a cephalad direction. The thoracic spinous processes point markedly downward, so that the distance between their tips and the corresponding segments of the spinal cord is greater than in the cervical region.

The lower thoracic and lumbar spines and laminae are relatively thick and short, the spines point directly backward and are deeply placed between thick muscles, so that the exposure of the dural sac by laminectomy is a more tedious procedure. Quite often, the thick short laminae have to be removed to the transverse and sometimes into the articular processes before sufficient exposure of the dura is obtained.

The Spinal Cord and Nerve Roots. The adult spinal cord is about 45 cm. long and extends from the margin of the foramen magnum to the lower part of the body of the first lumbar vertebra. At its lower end it tapers off conically, to end in a slender filament—the filum terminale. Grossly, the spinal cord ends at the first lumbar vertebra, but actually there is, in many instances, a certain amount of nerve tissue in at least the cephalad one-third of the filum terminale.

In the fetus, the cord extends to the lower end of the vertebral canal, but from the fourth month of postnatal life, the vertebral canal increases in length more rapidly than the spinal cord, so that at birth the tip of the conus lies at the level of the third lumbar vertebra. The changes in the relationship between canal and spinal cord and roots of the cauda equina after birth have an important bearing upon the disturbances produced by some congenital abnormalities of vertebrae and cord.

The cervical and lumbar enlargements of the spinal cord are almost entirely due to an expansion in a transverse direction. The cervical enlargement extends from the medulla to the level of the second thoracic vertebra, while the lumbar enlargement begins at about the tenth and is largest opposite the twelfth thoracic vertebra.

Somewhat arbitrarily, the spinal cord is divided into segments; each portion which corresponds to a pair of spinal roots is termed a segment, and the boundaries of each segment correspond to an imaginary horizontal plane through the cord midway between the origin of two adjacent pairs of roots. The first pair of cervical roots emerges from the vertebral canal between the occipital bone and the atlas; the upper seven cervical roots are named after the lower of the two vertebrae which form the intervertebral foramen for the exit of the root. The eighth cervical root emerges from the foramen between the seventh cervical and first thoracic vertebrae, and the thoracic, lumbar, and sacral roots are named after the upper of the two vertebrae which form the corresponding intervertebral foramen.

Each spinal nerve is formed by the junction of two roots which spring from the lateral aspects of the cord—the anterior or motor and the posterior or sensory root. The anterior and posterior roots are covered by extensions of the leptomeninges, and perforate the dural sheath separately with a thin septum of dura mater between them.

In the cervical region the nerve bundles remain distinct until they have passed through the dura, the bundles originating from the cord being spread out like a fan, the broadest part being at the cord. The dorsal and lumbar roots are formed by nerve bundles which soon unite to form the root which passes as one bundle to the dural opening.

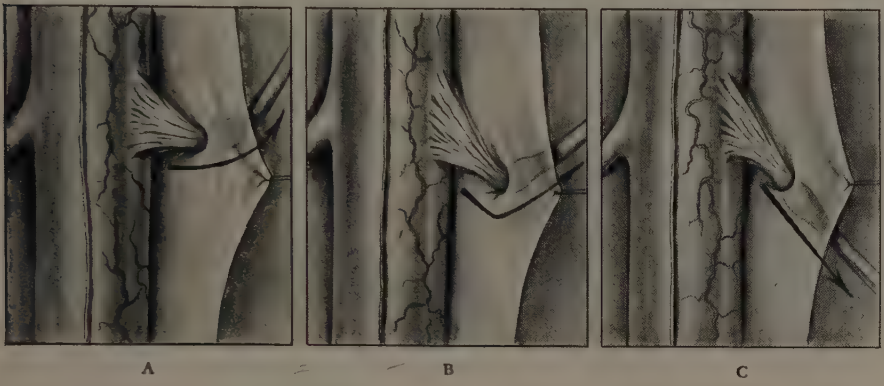


FIG. 1. The course of the posterior spinal roots in (A) the cervical region, (B) the thoracic region, and (C) the lumbar region of the cord (diagrammatic). (For explanation see text.)

From this arrangement it results that in the cervical region, a tumor may, for a time, make pressure upon only a few of the bundles which make up the root, while in the thoracic and lumbar regions, a tumor may, from the beginning, press upon the entire root.

There are marked differences between the course of the spinal roots at different levels (Fig. 1). The cervical roots pass out to the dural foramen almost at right angles to the spinal cord. Each root then passes through the dura and beyond the ganglion the nerve passes outward with a slight inclination upward.

The eighth cervical and the upper six to eight thoracic roots run in a somewhat different direction. Each root has an inclination downward until it nears the dura; it bends upward at an angle just as it passes through the dura. This angle is often one of 40 to 45 degrees. Beyond the ganglion the nerve passes markedly upward before dividing into anterior and posterior branches.

The lower dorsal and the lumbar and sacral roots pass downward and outward to the dura, and beyond the ganglia the direction remains almost unchanged until each nerve divides into anterior and posterior branches.

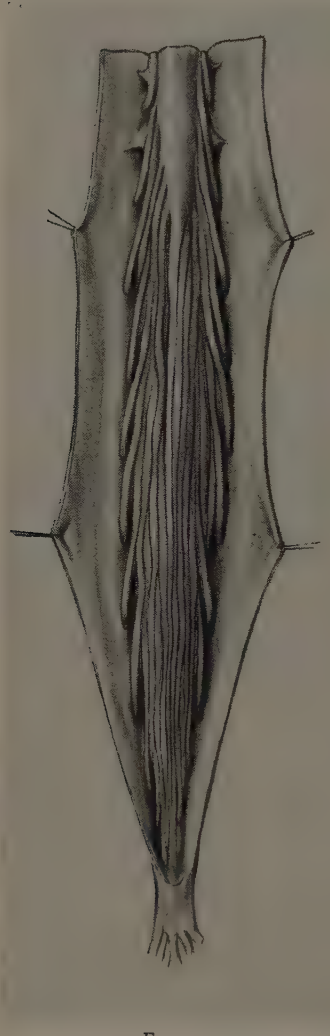


FIG. 2

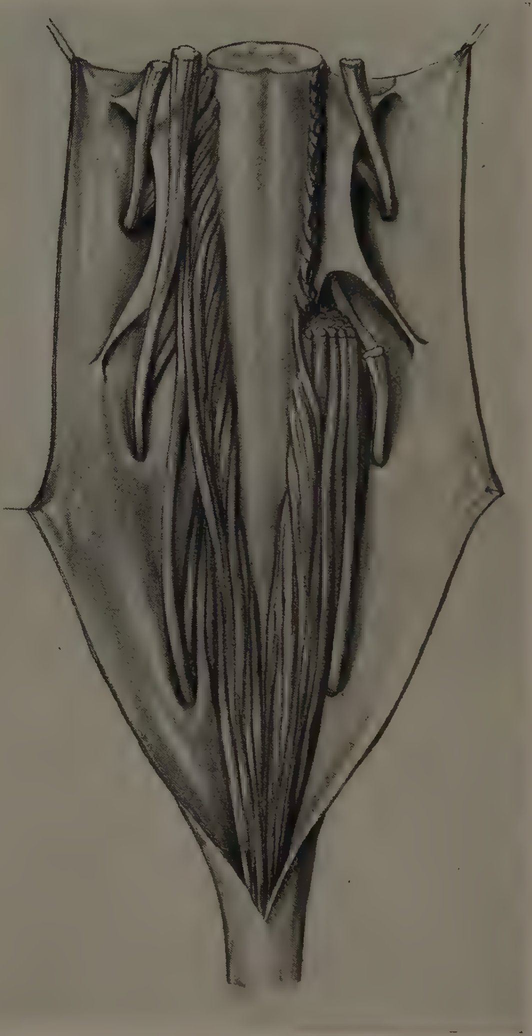


FIG. 3

FIG. 2. The arrangement of the roots of the cauda equina.
FIG. 3. The fork of the dentate ligament exposed on the left by division of some of the roots of the cauda equina on that side.

In general, the cervical roots have a course at almost a right angle to the long axis of the spinal cord. The lower the thoracic or lumbar level, however, the more oblique the course, so that, for example, the fifth lumbar roots emerge from the dural sac six vertebrae lower than the level of their origin from the spinal cord. The lumbar and sacral roots descend in almost parallel bundles to form the cauda equina and to conceal the filum terminale. The arrangement of the roots of the cauda equina is such that the outermost roots are the highest lumbar, and the most mesially situated roots are the lower sacral (Fig. 2).

From this arrangement it results that a tumor or other disease which involves the most mesially situated roots—the lower sacral roots—is most apt to produce early disturbances of the control of the bladder, as occurs, for example, in tumors of the filum terminale. More laterally situated lesions will disturb the functions of one or more of the upper lumbar roots, as, for example, in laterally placed tumors and in laterally situated herniations of a nucleus pulposus.

Because the roots of the cauda equina run so close to each other, disease in this region usually produces disturbances referable to a considerable number of the roots.

The Spinal Membranes. The spinal dura mater forms a loose sheath around the spinal cord and cauda equina, and is prolonged for a short distance over the spinal nerves. In the lumbosacral region, the dura is thinner than at higher levels.

The arachnoid is a thin transparent membrane which, normally, is in contact with but not adherent to the inner surface of the dura, but is connected with the pia mater by fine bands. Under normal conditions, the subarachnoid space is filled with fluid which acts as a buffer to support the spinal cord and to protect it from injury. The pia mater is intimately adherent to the spinal cord and forms its sheath.

From each lateral surface of the cord, derived from the pia mater, a narrow fibrous band, the *ligamentum denticulatum*, extends from the foramen magnum to the situation of the first lumbar root. From its attachment to the cord, each dentate ligament extends laterally, and is attached to the inner surface of the dura by numerous dentations or slips. The dentate ligament ends below at the general level of the first lumbar vertebra, in a fork-shaped extremity (Fig. 3).

As I pointed out many years ago, the outer prong of the fork is attached to the inner surface of the dura, while the inner prong of the fork is attached to the pia mater on the lateral aspect of the cord and is prolonged caudally along the side of the conus to its tip. Below the tip of the cord, the prolongation of the inner fork unites with its fellow of the opposite side to form the filum terminale.

The "fork" of the dentate ligament may be used as a surgical landmark for the identification of the first lumbar posterior root which rests upon the fork.

The dentate ligaments incompletely divide the subarachnoid space into anterior and posterior compartments. The dentate ligament is of considerable surgical significance, for it enables the surgeon to raise and rotate any part of the spinal cord without handling the cord directly. Furthermore, the classification of the relations of tumors to the different sides of the spinal cord is partly based upon their relation to the dentate ligament, and this structure may have an important bearing upon the occurrence of anterior root or posterior root symptoms before the appearance of disturbances due to pressure upon the cord itself.

The Relation of Cord Segments to Vertebrae (Fig. 4). As is well known, the segments of the cord do not lie on the same level as the correspondingly named vertebrae, and there are many variations in the relation between cord segments and vertebrae. In general, the lower boundary of the cervical cord (the level of the eighth cervical nerve) corresponds to the sixth cervical spinous process or the interspace between the fifth and sixth cervical spines. The fourth cervical segment lies about opposite the third cervical spinous process; the fourth thoracic, opposite the first or second thoracic spine; the eighth thoracic, opposite the fifth thoracic; the twelfth thoracic opposite the ninth thoracic; the second lumbar, at the level of the tenth thoracic; the fifth lumbar, at the level of the twelfth thoracic; and the sacral segments opposite the twelfth thoracic and first lumbar spines. The cord segments, therefore, lie at a higher (more cephalad) level than the correspondingly named vertebrae.

The relations of cord segments to vertebrae are of no little surgical significance. The localization of a lesion is mainly made from the highest level of motor and sensory disturbances and is ordinarily stated in terms of the segmentary level. From the surgical standpoint, however, the vertebral level is of importance, because the correct segment or segments will be exposed only when the proper vertebral arches are removed. The identifica-

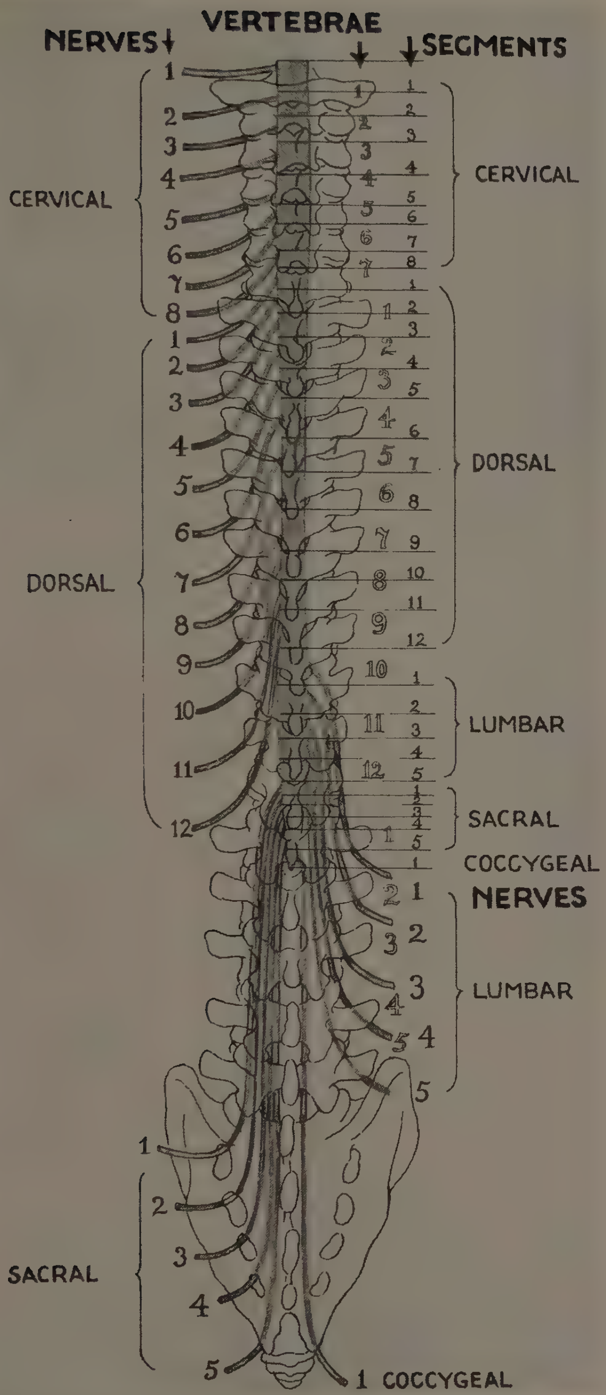


FIG. 4. The relation of the segments of the cord to the vertebrae (diagrammatic).

tion of the desired spinous processes by counting downward from the prominent seventh cervical spine and upward from the fourth lumbar spine (on a line connecting the highest parts of the iliac crests) is a satisfactory method, but in stout individuals it is not always reliable. The best method (see Chapter xv) is to mark a spot on the skin at the level of the desired spinous process and then, with a marker on the skin to have a roentgenogram of this part of the spine taken.

In recent years, the diagnosis of localized disease of the spinal cord or nerve roots has become easier due to the advances made in our knowledge of the changes in the vertebrae visible on roentgen films, to the information gained from tests of spinal subarachnoid block and examinations of the spinal fluid obtained by lumbar puncture, and to the use of lipiodol. These procedures should be used to *confirm* or *correct* the conclusions arrived at by clinical examination, but should not be used as short cuts to diagnosis. A well taken clinical history and a careful and thorough neurological examination must always be the firm foundation on which a diagnosis is made, and laboratory methods must be looked at as aids—and always important and necessary aids—to clinical findings.

The interpretation of symptoms and signs is based upon a knowledge of the gross anatomy of the spinal cord, membranes, and nerve roots, and their relations to the vertebrae of the course of fiber tracts, and upon an adequate understanding of the physiology and the localization of function in the various segments of the spinal cord. Most of this information can be found in textbooks of anatomy and physiology, and will not be given in detail here. However, there are some facts to which special attention may be directed.

Normally, there is, on all sides of the spinal cord, a space of about ½ cm. between the surface of the cord and the wall of the vertebral canal. The spinal cord, surrounded by the membranes, is suspended in the canal by the spinal roots and the slips of the dentate ligament, and has a certain mobility both in longitudinal and transverse directions. When this mobility is interfered with by a neoplasm or by an inflammatory process, an abnormal condition exists so that the normal compensatory movements of the cord with movements of the vertebrae are interfered with. As a result, movements of the spine may produce disturbances both in the fixed part of the cord and in the affected nerve roots, and unconsciously part of the spine is held rigid. Thus, in tumors and other lesions of the cervical cord, there is usually some stiffness of the neck and the normal curve of the cervical spine is more or less flattened out. Similarly, but often to a less degree, there is rigidity of the thoracic or lumbar vertebral column in diseases of the thoracic or lumbar cord. The stiffness of part of the spine is often a defense reaction on account of radicular pain.

During forced expiratory movements such as sneezing, coughing or straining, the mobility of the cord and the patency of the subarachnoid space is sufficient to prevent any radicular disturbances when cerebrospinal fluid is expelled from the cranial cavity into the spinal subarachnoid space. If, however, the cord is fixed by disease or the subarachnoid space is partially or completely obstructed, the mechanical conditions are altered, so that forced expiratory movements and the sudden passage of fluid into the spinal subarachnoid space result in sudden stretching of posterior spinal roots at the affected level and as a result, sudden pain in the back or pain of radicular distribution. When there is an expanding lesion in the spinal canal, the pain on forced expiratory movements may be due to the sudden impact of the tumor on a posterior spinal root.

A knowledge of the course of the fiber tracts is of no little importance for the recognition of pathological processes inside or outside of the spinal cord. Predominantly (but not entirely) the *posterior white columns* contain the fiber tracts that transmit the so-called deep sensibilities—vibratory and muscle-joint-tendon sensibility, and the like. These fibers run the entire length of the spinal cord in the posterior white column of one side, and in an affection which involves one posterior white column, vibratory, muscle-joint-tendon and the other "deep" sensibilities are disturbed on the same side as that of the lesion. The fibers from the sacral areas lie nearest the midline and most ventral, and the fibers from areas above the sacral take their position outside of and slightly posterior to the sacral fibers. Either because of the anatomical arrangement or on account of different degrees of sensitivity of the fibers in the posterior white columns, vibratory and muscle-joint-tendon sensibility are not invariably equally disturbed. Vibratory sensibility may be diminished or lost, while muscle-joint-tendon sense is preserved, or vice versa. Without doubt, fibers which transmit cutaneous sensibility also run in the posterior white columns.

The *anterolateral white columns* transmit the pyramidal and the lateral spinothalamic tracts as well as some fibers that have to do with tactile sensibility, and perhaps fibers that influence the function of the bladder and rectum. The pyramidal fibers, after their decussation in the medulla, run in a downward direction in one lateral column, so that a lesion which involves these pathways on one side, causes changes in power and in the reflexes of the same side. Interference with the pyramidal fibers causes a diminution in power and hyperactivity of tendon reflexes on the same side as a unilateral lesion. There is some probability, although the evidence is by no means conclusive, that in the pyramidal pathways, the fibers which supply the muscles of the limbs (and of parts of limbs) have a lamellated arrangement—the fibers which supply muscles at a higher level lying more mesial than those supplying muscles at a lower level.

The *main spinothalamic pathways* are in the anterolateral white columns in which they lie ventral to the pyramidal tracts. They carry fibers which transmit cutaneous sensibility for pain and for temperature in a cephalad direction. After entering the spinal cord, these fibers soon cross the midline to take their position in the spinothalamic tract of the opposite side. It was formerly believed that the crossing required two or more segments of the spinal cord, but, from the investigations of Foerster and others, it appears probable that, in the majority of instances, the crossing occurs in one cord segment.

For many years, the belief was prevalent that there was not a lamellation of the fibers in the spinothalamic pathways. The clinical investigations of Foerster, the writer, and others have made it certain that, in these pathways, the fibers are arranged so that the fibers which convey sensation from the most caudal skin areas lie in the posterior and outer part of each tract, and those which join at higher levels take a position somewhat anterior and *internal* to the ones already in the tract (Fig. 5). In extramedullary tumors, the history often reveals that the subjective sensory disturbances began in the most distal parts of the lower extremities and gradually advanced in a cephalad direction; and, on the other hand, in intramedullary growths, that diminished sensibility was first observed on the chest or abdomen and then spread in a caudal direction. In the early stages of compression of the spinal cord by an extramedullary tumor which lies dorsally or dorsolaterally, only the most external and posterior parts of the lateral spinothalamic tracts may be affected by the pressure; and disturbances of sensibility may be found only in dermatomes well below the actual segmentary level of the lesion. If all cutaneous sensibility has been disturbed below the cord segment compressed by the growth, the greatest diminution of sensation will often be found in the most caudal dermatomic areas. In the early stages of cord compression by an extramedullary tumor on the median ventral aspect of the cord, sensibility is often undisturbed in the most caudal dermatomes which means that at first, the pressure of the expanding

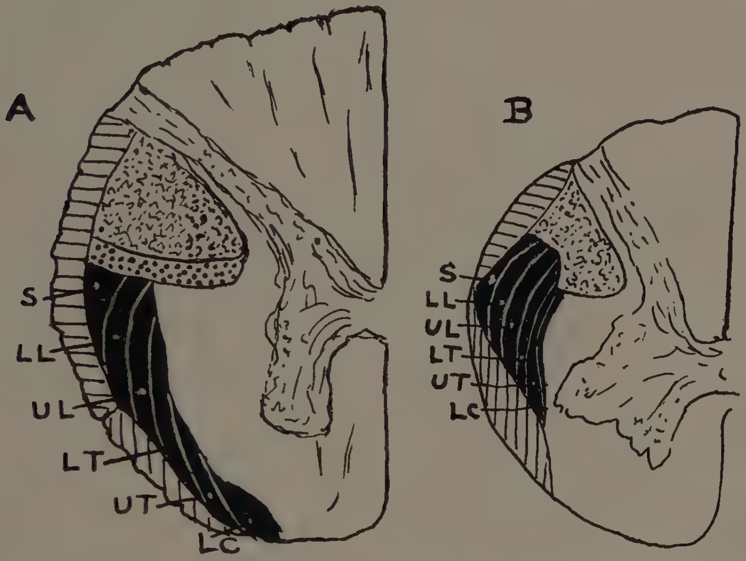


FIG. 5. The possible lamellar arrangement of the fibers in the lateral spinothalamic tracts. *S,* sacral; *LL,* low lumbar; *UL,* upper lumbar; *LT,* lower thoracic; *UT,* upper thoracic; *LC,* lower cervical. The location and size of the spinothalamic tract in A is from Tilney and Riley, and in B from Déjèrine (diagrammatic).

lesion was transmitted only to the most anterior and internal parts of the lateral spinothalamic tracts. On the other hand, in pressure upon the cord from behind by a small tumor near or in the midline, such a relative sparing of fibers from the more caudal skin areas is never seen. Many cases of extramedullary compression of the spinal cord have been described in which the sacral dermatomes were not involved or in which the disturbances of sensibility were in areas well below the segmentary level.

Likewise, it is well known that after division of the anterolateral tracts for pain, the upper level of loss of pain and temperature sensibility occurs in dermatomes far below the segment at which the cord has been incised. In these cases it is probable that the incision in the cord was not deep enough to divide the innermost and most anterior parts of the spinothalamic tracts.

Much additional clinical evidence could be cited to support the belief that there is an exquisite lamellation of fibers in the spinothalamic pathways.

The evidence that the fibers for pain and for temperature sensibility run in separate pathways in the spinothalamic tracts is not conclusive. Both from clinical studies of spinal cord diseases and from the results of partial division of the spinothalamic tracts, there is much to support that view. The clinical evidence which points in that direction has been summarized in the chapter on traumatic lesions of the spinal cord.

Clinically, pain and temperature sensibility are not always equally affected in tumors and other diseases of the spinal cord. Not rarely, the ability to recognize warmth and cold is preserved in areas in which pain sensibility is lost, and vice versa. Likewise, the ability to distinguish warmth and cold may be dissociated, moderate degrees of cold being recognized when warmth is not appreciated as such. For reasons not yet well understood, there is, especially in some instances of intramedullary disease, a marked delay in the ability to recognize cold on the affected as compared with the normal side.

In complete transverse lesions of the spinal cord at any level (*q.v.*), the upper level of pain and temperature disturbances may not coincide with that of the loss of tactile sensibility—the upper level of the former being lower or higher than that of the latter. This difference may be due to various factors. The level for touch may be higher because an area of radicular loss may be above and added to that due to the spinal cord lesion. Furthermore, because of the fact first pointed out by Sherrington, that dermatomes may be supplied by three spinal nerve roots, the upper level of cutaneous disturbances for touch may be at a lower level than that for pain and temperature. When, on the other hand, disturbances of pain and temperature sensibility have a higher level than that of tactile sensibility, there is the suspicion that an intramedullary lesion (such as hematomyelia in complete transverse lesions due to trauma) extends more cephalad than the main lesion of the spinal cord. However, some of the differences just mentioned may be explained by the methods used for the tests of the various modalities of cutaneous sensibility, by the varying degrees of stimulation of the peripheral receptors necessary for the starting of a nervous impulse and the variations in the sensitiveness of the central areas which have to do with conscious perception and recognition of the senses of touch, pain, and temperature.

The situation of the pathways in the spinal cord which carry afferent fibers from the bladder and rectum, and which control reflex and voluntary vesical and rectal evacuation, are uncertain. There is some evidence to support the belief that they run a juxtagriseal course and other evidence that their course is in the gray matter of the cord. The bladder mechanism is a complicated one in which autonomic nerves play an important part. For detailed discussion of this subject, the reader is referred to the textbooks of Fulton, Brock, and others.

It is beyond the scope of this chapter, to enter into a discussion of the anatomy and physiology of the various segments of the spinal cord, of the cells in the anterior horns which are the spinal centers for the muscles of the limbs and trunk, and for the various tendon and cutaneous reflexes. Tables I and II showing the segmentary localization of the muscles and of the spinal centers for the skin and tendon reflexes and the chart showing the dermatomes (Fig. 6) may be useful.

Table I has been modified from the tables of Flatau and of Foerster, to which I have added the main function of each muscle or muscle group.

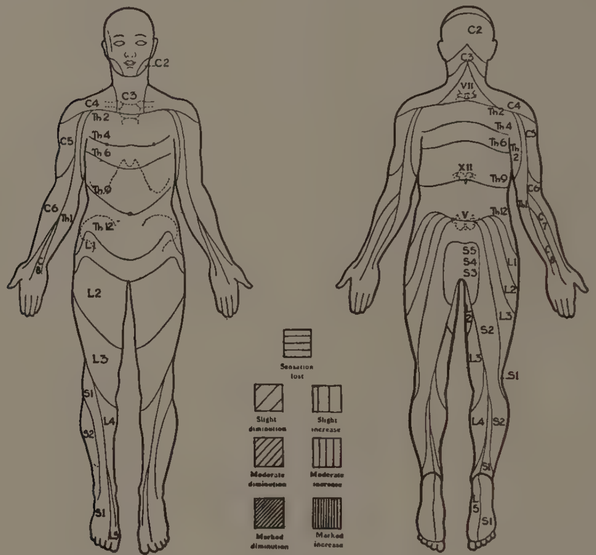


FIG. 6. Diagrammatic chart showing the general limits of the dermatomes. The limits shown are not exact (see text).

TABLE I

THE SEGMENTARY LOCALIZATION OF THE MUSCLES

A. Muscles of the Head and Trunk

I. Muscles of the Back		
Trapezius	C 1–C 4	Rotation of scapula, raises shoulder, moves head to side
Latissimus dorsi	C 6–C 8	Adducts and rotates arm inward, draws arm backward, raises ribs
Rhomboid	C 4–C 5	Rotates and moves scapula backward
Levator anguli scapulae	C 3–C 5	Raises angle of scapula
Rectus capitis major	C 1 C 2	Rotate head and draw it back
Rectus capitis minor	C 1 C 2	
Obliquus capitis superior	C 1 C 2	Rotate head and draw it back
Obliquus capitis inferior	C 1 C 2	
II. Muscles of Neck, Chest, and Abdomen		
(a) Muscles of the neck:		
Platysma	C 3	Depresses lower lips and angle of mouth, wrinkles skin of neck
Sternomastoid	C 1–C 3	Rotates and draws head to shoulder, muscle of inspiration

TABLE I (*Continued*)

Sternohyoid	C1 C2 C3	Depress larynx and hyoid bone, control movements of thyroid cartilage
Omohyoid	C1 C2	
Sternothyroid	C1 C4	
Thyrohyoid	C1 C2	
Longus capitis	C1 C4	Flexes and rotates cervical vertebral column
Rectus capitis anticus	C1	Flexes and rotates head
Scalenus anticus	C4–C8	Raise ribs for inspiration
Scalenus medius	C4–C8	
Scalenus posticus	C4–C8	
(b) Muscles of the chest:		
Pectoralis major	C5–C8 Th1	Adduction, downward and forward movement of arm
Pectoralis minor	C6–C8	
Subclavius	C5 C6	Depresses shoulder
Serratus anticus	C5–C7 (C8)	Fixes scapula
Levatores costarum	Th1 Th12	Fix ribs
Intercostals	Th1 Th12	Inspiration
Diaphragm	(C2) C3–C5	Respiration
(c) Muscles of the abdomen:		
Rectus abdominis	Th5–L1	Constrict cavity of abdomen, assist expiration, rotate trunk, move pelvis
Pyramidalis	Th5–L1	
Obliquus externus	Th5–L1	
Obliquus internus	Th5–L1	
Quadratus lumborum	Th12 L1 L2 L3	Moves pelvis and trunk, inspiration

B. Muscles of the Extremities

I. Muscles of the Upper Extremities

(a) Shoulder:		
Deltoid	C5 C6	Abduct arm to horizontal, abduct and rotate arm outward
Supraspinatus	C4 C5	
Infraspinatus	C4–C6	
Teres minor	C4–C6	Rotates arm outward
Teres major	C6–C7	Rotates arm inward
Subscapularis	C6–C8	Rotates arm inward
(b) Arm:		
Biceps	C5–C6	Flexes and supinates forearm
Coracobrachialis	C6–C7 (C8)	Adducts forearm
Brachialis anticus	C5–C6	Flexes forearm
Triceps	C7 C8 Th1	Extends forearm
(c) Forearm:		
Pronator radii teres	C6–C7	Pronates forearm
Flexor carpi radialis	(C6) C7–C8	Flexes and radially flexes hand
Palmaris longus	(C7) C8 Th1	Flexes hand
Flexor carpi ulnaris	(C7) C8 Th1	Flexes and ulnar flexes hand
Flexor sublimis digitorum	(C7) C8 Th1	Flexes middle phalanges, 2–5 fingers
Flexor profundus digitorum	C7 C8 Th1	Flexes last phalanges, 2–5 fingers
Flexor longus pollicis	C8 Th1	Flexes last phalanx of thumb
Pronator quadratus	C8–Th1	Pronates forearm
Extensor carpi radialis	C6–C7	Extends radially, radially flexes hand
Extensor communis digitorum	C6 C7 C8	Extension of first phalanges, 2–5 fingers
Extensor minimi digiti	(C6) C7 C8	Extension of first phalanx of little finger
Extensor carpi ulnaris	C7 C8	Extension and ulnar flexion of hand
Supinator brevis	C5 C6	Supinates forearm
Abductor longus pollicis	(C6) C7 C8	Abducts first metacarpal
Extensor brevis pollicis	(C7) C8 Th1	Extension of first phalanx of thumb
Extensor longus pollicis	(C7) C8 Th1	Abducts first metacarpal; extension of last phalanx of thumb

Table I (*Continued*)

Extensor proprius indicis	(C7) C8 Th 1	Extension of first phalanx of index finger
(d) Hand:		
Abductor brevis pollicis	C7 C8	Abducts first metacarpal
Flexor brevis pollicis	C7 C8	Flexes first phalanx of thumb
Opponens pollicis	C7 C8	Opposition of first metacarpal
Adductor pollicis	C7 C8	Adducts first metacarpal
Abductor minimi digiti	C8 Th 1	Abducts little finger
Flexor brevis minimi digiti	C8 Th 1	Abducts and flexes little finger
Lumbricales	C8 Th 1	Abduct and adduct fingers
Interossei	C8 Th 1	Extension of 2nd and 3rd phalanges
II. Muscles of the Lower Extremities		
(a) Hip:		
Iliopsoas	L1 L2 L3 L4	Flexion at hip
Gluteus maximus	L5 S1 S2	Extension of thigh
Tensor fasciae latae	L4 L5	Flexion of thigh
Gluteus medius	L4 L5 S1	Abduction and internal rotation of thigh
Gluteus minimus	L4 L5 S1	
Obturator internus	L4 L5 S1 S2	External rotation of thigh
Quadratus femoris	L4 L5 S1 S2	
(b) Thigh:		
Sartorius	L1 L2 L3	Internal rotation of leg
Rectus femoris	L2 L3 L4	Extension of leg
Vastus medius	L2 L3 L4	
Vastus internus	L2 L3 L4	
Vastus externus	L2 L3 L4	
Pectineus	L2 L3 L4	
Adductor longus	L2 L3 L4	
Gracilis	L2 L3 L4	Adduct thigh
Adductor brevis	L2 L3 L4	
Adductor magnus	L2 L3 L4	
Obturator externus	L3–L4	Adduction and external rotation of thigh
Biceps	L5 S1 S2	Flex leg
Semitendinosus	L5 S1 S2	
Semimembranosus	L5 S1 S2	
(c) Leg:		
Tibialis anticus	L4 L5	Dorsal flexion and supination of foot
Extensor longus digitorum	(L4) L5 S1	Extension of toes
Peroneus tertius	L5 (S1)	Dorsal flexion and pronation of foot
Extensor longus pollicis	(L4) L5 S1	Extension of large toe
Peroneus longus	L5 S1 S2	Dorsal flexion and pronation of foot
Peroneus brevis	L5 S1 S2	
Gastrocnemius	(L5) S1 S2	Plantar flexion of foot
Popliteus	L5 S1 S2	Flexion of leg
Tibialis posticus	L4 L5 (S1)	Adduction of foot
Flexor longus digitorum	(L5) S1 S2	Flexion of last phalanges II to V
Flexor longus pollicis	(L5) S1 S2	Flexion of last phalanx of large toe
(d) Foot:		
Extensor brevis pollicis	(L5) S1 S2	Extension of large toe
Extensor brevis digitorum	(L5) S1 S2	Extension of toes
Abductor pollicis	S1 S2 S3	Movement of toes
Flexor brevis pollicis	S1 S2 S3	
Adductor pollicis	S1 S2 S3	
Abductor minimi digiti	S1 S2 S3	
Opponens minimi digiti	S1 S2 S3	
Flexor brevis digitorum	S1 S2 S3	
Lumbricales	S1 S2 S3	
Interossei	S1 S2 S3	

TABLE II

THE CENTERS FOR THE SKIN AND TENDON REFLEXES IN THE SPINAL CORD

(Modified from Bing)

Tendon reflex	*Skin reflex*	*Elicited by*	*Result*	*Localization*
	Scapula reflex	Irritation of skin over scapula	Contraction of muscles of shoulder blade	C 5–Th 1
Biceps reflex	....	Blow on tendon of biceps	Flexion of forearm	C 5–C 6
Triceps reflex	....	Blow on tendon of triceps	Extension of forearm	C 7–C 8
Scapulohumeral reflex	....	Blow on lower inner angle of scapula	Adduction of arm	C 6–C 7
Radius reflex	....	Blow on styloid process of radius	Supination of forearm	C 7–C 8 (C 5–C 6?)
	Palmar reflex	Irritation of palm of hand	Flexion of fingers	C 8–Th 1
	Epigastric reflex	Irritation of lower part of chest	Contraction in epigastric region	Th 7–Th 9
	Upper abdominal reflex	Irritation of skin over upper abdomen	Contraction of abdominal muscles	Th 8–Th 9
	Lower abdominal reflex	Irritation of skin of lower abdomen	Contraction of abdominal muscles	Th 10–Th 12
	Cremaster reflex	Irritation of skin over inner surface of thigh	Elevation of testis	L 1–L 2
Patellar reflex	....	Blow on tendon of quadriceps	Extension of leg	L 2–L 4
	Gluteal reflex	Irritation of skin of gluteal region	Contraction of gluteal muscles	L 4–L 5 (S 1)
Achilles reflex	....	Blow on Achilles tendon	Extension of foot	S 1–S 2
	Plantar reflex	Irritation of skin of sole	Flexion of toes	S 1–S 2
	Anal reflex	Pricking of perineum	Contraction of sphincter ani	S 5

II. DIAGNOSIS OF LOCALIZED SPINAL DISEASES IN WHICH SURGICAL PROCEDURES MAY BE NECESSARY

IN OTHER CHAPTERS DEALING WITH INJURIES, TUMORS, AND OTHER LESIONS OF THE SPINAL CORD AND MEMBRANES, THE CRITERIA ON which diagnosis and localization must be based, will be spoken of. In many instances, the recognition that an individual is suffering from a localized disease of the spinal cord in which surgical intervention is indicated, is not difficult if the progression of symptoms is characteristic, the objective neurological disturbances point to a definite level, and manometric tests and alterations of the fluid withdrawn by lumbar puncture (and perhaps roentgenograms of the spine) confirm the conclusion arrived at from the examination.

However, many cases are not as simple as this and variations in the clinical course and in the disturbances found at the examination are frequent.

An acute onset of spinal cord disturbances occurs almost exclusively in inflammatory lesions, such as extradural abscess, or in metastatic growths. Such an acute onset, with a history dating back only a few weeks or a few months, is extremely rare in extramedullary tumors; but vague symptoms may become definite after lumbar puncture and withdrawal of spinal fluid.

For example, I have operated upon and removed a tumor at the seventh cervical level from a patient who had only a slight weakness of one lower limb without subjective or objective sensory disturbances, and in whom marked motor and sensory loss up to a definite level followed within twelve hours of lumbar puncture and withdrawal of fluid.

When the disease is localized in a definite segment and one side of the spinal cord and there is a progression of symptoms, the order in which the disturbances appear usually depends upon the mechanical changes that are occurring. Thus, a tumor which first involves a sensory spinal root will at first irritate and then interfere with the functions of that root; when the growth has increased in size sufficiently to make pressure upon the spinal cord, the homolateral side of the cord will first be affected, so that motor and deep sensibility disturbances on the same side, and cutaneous disturbances of pain and temperature sensibility on the opposite side of the body will first occur and may be observed subjectively by the patient and be found objectively by the physician. Such a progression of symptoms and signs is characteristic of a tumor or some other expanding extramedullary disease. The rate at which the symptoms and signs advance depends upon the situation of the disease, the rapidity with which the lesion increases in size and its nature, but in the majority of instances many months, and not so rarely years, pass before the patient is seen by the neurologist and surgeon.

If the lesion lies ventral to the spinal cord, pain may not be complained of for a long period, and motor weakness or atrophy due to anterior root involvement may be so slight that it may be missed altogether. As regards pain, there is a characteristic difference between tumors that lie on the ventral aspect of a spinal cord segment and growths or other lesions (spinal gliosis, syringomyelia, etc.) within the substance of the spinal cord. In both ventral extramedullary and intramedullary lesions pain is rare as an early symptom, but in the ventral extramedullary lesion it will occur sooner or later, while in intramedullary disease there may never be any pain.

The pain may be felt in the back or it may radiate around a part of the trunk or into one limb. In an upper extremity, it may have the character of a brachial neuralgia; in the chest, it may be like that caused by pleuritic adhesions; in the abdomen, it may be like that of intra-abdominal disease; and in a lower limb, it may simulate sciatica.

Pain on pressure over the back is frequent in spinal cord lesions of various kinds. The tenderness of one or more spinous processes on pressure or percussion is well known. This tenderness may be a true bone tenderness due to an osseous lesion, or may be a true posterior spinal root tenderness. Thus, in compression of the spinal cord and nerve roots by tumor, the tender spinous process is usually one or two vertebrae below the actual vertebral level of the lesion, while true bone tenderness is at the real level of the bone disease.

The order in which motor (and sensory disturbances) occur is much more regular in extramedullary spinal cord tumors than in other diseases outside of or within the substance of the spinal cord.

It may be considered a general rule that the more posteriorly an expanding lesion is situated outside of the cord, the more frequently are motor (and also sensory) disturbances first observed in the most distal parts of the lower limbs. This may be explained by the lamellation of the spinothalamic and, probably, the pyramidal tracts.

Unless the lesion involves the anterior horns in a segment which has to do with the reflex activity of any muscle or group of muscles, the tendon reflexes are always hyperactive below the level of the lesion, either on one or both sides. If the tendon reflexes of both sides are hyperactive, the most marked change is usually on the side of the lesion. This is the case only if the compression of the cord from the outside or from a lesion within the substance of the cord is of a moderate degree of intensity. When the involvement of the cord is greater, there may be a stage where the tendon reflexes of one lower extremity are less or have been lost altogether, while those of the other side are still hyperactive. When this has occurred, the tendon reflexes may not be hyperactive in the limb in which the reflex disturbances are most advanced, and may be hyperactive in the limb which is less affected. The general rule, however, is that the tendon reflexes are more active on the side of the lesion, excepting in lumbosacral cord or cauda equina lesions. Here patellar and Achilles reflexes may be lost on the side of the lesion while they are normally active on the other side.

The loss of one Achilles reflex (with or without weakness in dorsal extension at the ankle) usually indicates a lesion of the lumbosacral cord or of the cauda equina, but it must be remembered that in lesions of the sciatic nerve, such as the clinical syndrome known as sciatica, the Achilles reflex may be lost. It is sometimes uncertain whether the loss of an Achilles reflex is due to spinal root or peripheral nerve involvement, even when a careful examination with spinal manometric tests and examination of the fluid obtained by lumbar puncture fails to show any evidence of an affection of the spinal cord or nerve roots.

The diminution or loss of one or other tendon reflex in one of the upper limbs, especially when there is associated with the loss, a hyperactivity of the tendon reflexes whose spinal centers lie in segments below the level, is often an exquisite localizing sign. Thus, diminution or loss of the biceps with hyperactive triceps reflex occurs with a lesion affecting the fifth and sixth cervical segments on the same side. In affections of the seventh and eighth cervical segments, there is often an inversion of the triceps reflex; that is, on tapping the triceps tendon, there is flexion instead of extension of the arm at the elbow. Weakness of flexor power at the elbow with normal extensor power, or weakness of extensor power with normal flexor power, when associated with the reflex changes mentioned, is of considerable significance. When comparing the power of the two upper limbs, however, it is well to remember that normally, in right-handed individuals, the power of the right upper limb is often distinctly greater than that of its fellow on the opposite side.

The diagnostic and localizing significance of changes in cutaneous and deep sensibilities will be considered in detail in Chapter IX. In extramedullary diseases, the subjective sensory disturbances often begin in the most peripheral parts of the limbs; in intramedullary diseases, the disturbances may begin near the segmentary level of the lesion. The neurological examination, if carefully made, will often show also, that the objective sensory disturbances are more marked in the caudal parts of the lower limbs in extramedullary, and nearest the level of the lesion in intramedullary disease. However, there are many exceptions to this rule; the most important one is that in ventral extramedullary lesions the sensory disturbances may be very like those in intramedullary disease.

Dissociation of cutaneous sensibility—relative sparing of tactile as compared to pain and temperature sensibility—is more characteristic of disease within the substance of the spinal cord, but such dissociation is not rare in ventral extramedullary expanding lesions and in many instances of compression of the cervical cord.

When there is a complete or a very marked involvement of sensation, the determination of the exact upper level of sensory disturbance, and therefore of the localization of the disease, is usually easy. On the other hand, in the early stages of spinal cord compression, especially in the thoracic region, it may be difficult to map out the true upper level of diminished cutaneous sensibility, because the disturbance of sensibility may gradually shade off in an upward direction. Furthermore, in the early stages of compression of the spinal cord, the pressure upon the cord may affect only those fibers in the spinothalamic pathways that transmit sensation from more caudal dermatomes. In such an individual, *especially if there have not been any root disturbances*, the apparent level of the lesion may be a considerable distance below the actual level.

This has sometimes led to operations at too low a level. In a patient upon whom I operated many years ago, there were clear level signs at the eleventh thoracic segment. The surgical intervention failed to disclose the tumor. One year later, there were clear indications of a lesion at the fourth thoracic segment. At a second operation, I found and removed an extramedullary growth at that level.

The diagnosis of a localized disease of the spinal cord and the locus of the disease is usually possible, if all the facts are carefully marshalled and correctly interpreted. However, it is not unusual to see patients in whom two distinct levels of disturbances of sensibility are made out—the one many segments above the other. Likewise, there may be a discrepancy between bony abnormality seen on the roentgen films and the level indicated by the examination. This latter condition of affairs is sometimes found in varicosities of the spinal veins (*q. v.*) in which definite changes in pedicles may be found in the x-ray films while the disturbances of sensibility point to a higher level.

The discrepancy is probably to be explained by the fact that the abnormality of the spinal veins has caused a change in the pedicles at a lower level than the most marked intrinsic cord changes.

In the majority of instances in which two sensory levels are found, there is either only one localized lesion, or the disease is not a strictly localized one (as in varicose veins of the spinal cord). Multiple metastases, and multiple tumor masses, such as occur in multiple myeloma, chordoma or Hodgkin's disease, are, all in all, quite unusual.

Sometimes the disease does not follow its usual course, and the symptoms referable to the spinal cord reach a certain degree of severity and then for years advance no further. Such a course is very unusual in extramedullary tumors; it is more frequent in very slowly progressive intramedullary disease, such as multiple sclerosis and spinal gliosis. There are without doubt other intrinsic diseases of the spinal cord whose nature is not well understood, and which exhibit marked motor and reflex disturbances and ill-defined diminution of cutaneous sensibility. In these cases the question has to be answered whether the patient is suffering from a localized disease which may be benefited by surgery or a disease in which surgery is not indicated. During the course of years, I have seen not a few cases of this kind, and I confess that in some of them I have performed an exploratory operation and found no evidence of a localized lesion. In the present state of knowledge, with the information to be gained by spinal manometric tests and the use of lipiodol, such operations can and should be avoided.

In a chapter devoted to the diagnosis of spinal cord disease, there is one condition which occurs after trauma to the back, to which attention must be directed. During the course of years, I have seen a not inconsiderable number of individuals who had been suspected of having some lesion of the spinal cord, because after a trauma to the back, they had developed more or less marked paralysis of the lower limbs with or without disturbances of sensibility. In some of these individuals a lesion of the spinal cord was suspected for which the patients were treated for many months and, in one instance at least, for several years. In these individuals there was a striking absence of objective neurological disturbances, excepting those that could be explained by disuse of muscles, and often a definite advance of purely subjective disturbance each time a new consultant examined the patient. Although a diagnosis of traumatic hysteria should be the last diagnosis that should be made in any patient with symptoms referable to the spinal cord, the physician should remember that such cases do occur. No harm can be done, in such a case, by the use of psychotherapy and in my experience I have seen not a few individuals quickly freed from disturbances which had existed for many months by a properly applied type of therapy of which the relatives were aware, but the *patient was not aware.*

CHAPTER II

LUMBAR PUNCTURE AND THE EFFECTS OF THE WITHDRAWAL OF CEREBROSPINAL FLUID

LUMBAR PUNCTURE IS ORDINARILY PERFORMED BELOW THE FOURTH LUMBAR SPINE, WHICH IS ON A LEVEL WITH THE HIGHEST PART OF the iliac crests. In the recumbent individual, a pressure between 60 and 120 mm. of water is usual, but a pressure up to 180 mm. or more may be entirely within normal limits.

If the mechanical conditions within the vertebral canal are normal and there is no pathological process in or around the spinal cord, membranes and spinal roots, the spinal fluid pressure is mainly the result of transmitted intracranial pressure. If, on the other hand, the canal is locally narrowed, due to changes in the bony framework of the spine, to enlargement of the spinal cord, or to the presence of a space occupying lesion such as a tumor, the pressure of the fluid is altered. A similar condition obtains when there are adhesions between the pia mater and the arachnoid. In each instance, and over a more or less localized area, the subarachnoid space is narrowed so that there is a more or less marked interference with the free passage of cerebrospinal fluid.

In most of the instances in normal individuals in which fluid does not escape through a lumbar puncture needle which has been introduced into the spinal canal, the needle either has not penetrated the subarachnoid space or the escape of fluid is prevented by nerve roots or other soft tissues that obstruct the lumen of the puncture needle. In certain cases of complete obliteration of the subarachnoid space at any level, either by adhesive processes between the leptomeninges or by a large neoplasm, only a small quantity of fluid can be obtained, after which the tap is a dry one.

In the individual in whom there is free communication between the cranial and spinal subarachnoid space down to the level of the lumbar puncture needle, the removal of fluid by lumbar puncture is often followed by headache which may last for one or for a number of days. When there is a complete or almost complete spinal subarachnoid block, lumbar puncture headache is never observed. Aside from this puncture headache, there are no other disturbances in healthy individuals. But in those suffering from a spinal lesion, a number of symptoms may develop after the removal of spinal fluid. Thus, root pains may become exaggerated (Elsberg, 1925; Viets, 1928; and others) or neurological disturbances may become aggravated. This is most often observed with extradural tumors of the spinal cord and with intradural growths that are adherent to the inner surface of the dura or to the outer surface of the arachnoid. In these instances, there may be a buffer of fluid between the growth and the spinal cord; when fluid is removed by lumbar puncture, the growth may fall against the cord and the existing spinal cord disturbances become more marked. The removal of fluid may result in more definite sensory signs and the upper level of diminution of sensation may become clearer. It is of some interest that a number of authors, among them Oppenheim (1907), Nonne (1913), Newmark (1914), and Antoni (1920), have spoken of the dangers of lumbar puncture and the aggravation of symptoms that may result from the withdrawal of cerebrospinal fluid. The significance of the changes that occurred seems to have impressed only Newmark, and the diagnostic value of the procedure was not noticed until after the publication of one of my papers (1922).

MANOMETRIC TESTS OF THE PATENCY OF THE PATHWAYS FOR FLUID IN THE SPINAL SUBARACHNOID SPACE

Many studies have been made, in both normal individuals and patients with spinal disease, of the pressure with which the spinal fluid escapes from the spinal puncture needle and the effect of pressure on the jugular veins.

The presence of a pathological process in the spinal canal which interferes with the free flow of cerebrospinal fluid from the cranial cavity into the spinal canal, is tested for by procedures in which the intracranial pressure is artificially raised so that the fluid escapes from a needle in the lumbar subarachnoid space under increased pressure. This is accomplished by light or firm digital compression of the jugular veins in the neck (Queckenstedt, 1916; Stookey and others) or by the inhalation of nitrite of amyl (Elsberg and Hare, 1935).

Grant and Cone have recommended graduated jugular compression by means of a blood pressure cuff. They believe that their method offers the following advantages: (a) a measured graduated jugular compression which can be exactly duplicated in the same patient; (b) a standard which can be used from patient to patient; (c) more dependability in the results obtained by different operators; and (d) automatic and simultaneous recording and timing on a kymograph of spinal fluid pressures. For general clinical use, the jugular compression and amyl nitrite tests are sufficient to demonstrate various degrees of spinal subarachnoid block, but more refined methods, such as that devised by Grant and Cone, may give valuable information.

In our clinic at the New York Neurological Institute we are accustomed to make both jugular compression and amyl nitrite tests. The two tests are usually mutually confirmatory but sometimes the nitrite of amyl test will demonstrate a block not shown by the jugular compression.

The compression of the jugular veins causes a venous congestion within the cranial cavity and, as a result, the expulsion of cerebrospinal fluid for the ventricular cavities. In the amyl nitrite test the same result is accomplished by the dilatation of the intracranial vessels which follows inhalation of the drug.

The *complete* manometric test by compression of the jugular veins should include a record upon an appropriate chart of (a) the effect of momentary light or "touch" compression of the jugular veins; (b) the effect of firm or "deep" compression for ten seconds; (c) the time required for the rise of the column of fluid to its high-

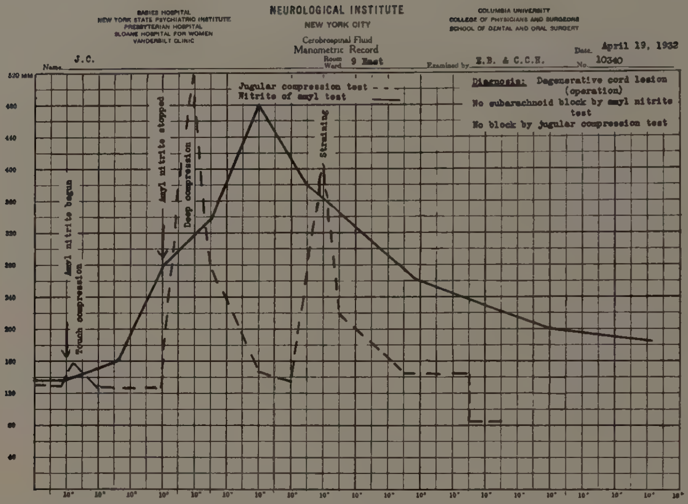


FIG. 7. Manometric chart showing curve characteristic of an unobstructed spinal subarachnoid space. The continuous line on this and the following charts is the curve obtained by the nitrite of amyl test, and the broken line, the curve obtained by the jugular compression test made as a control. The arrows which point downward indicate when the inhalation of nitrite of amyl was begun and when it was discontinued. The time period between the two arrows is thirty seconds.

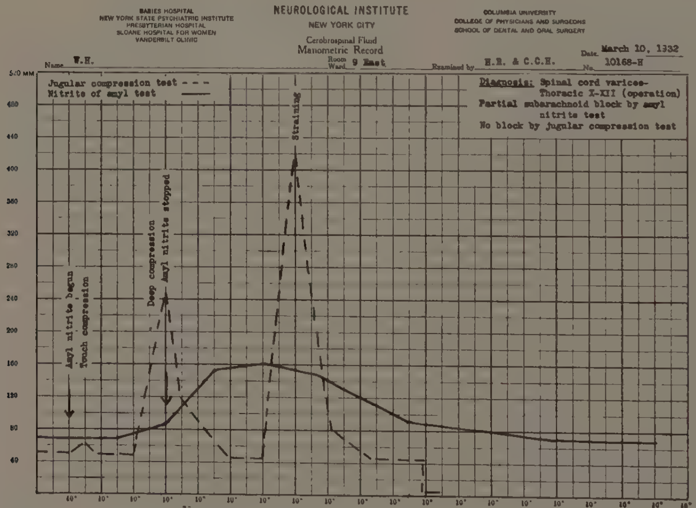
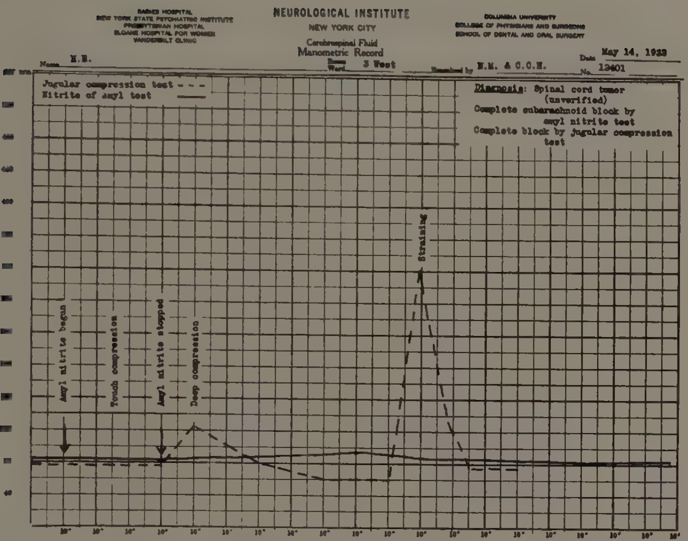


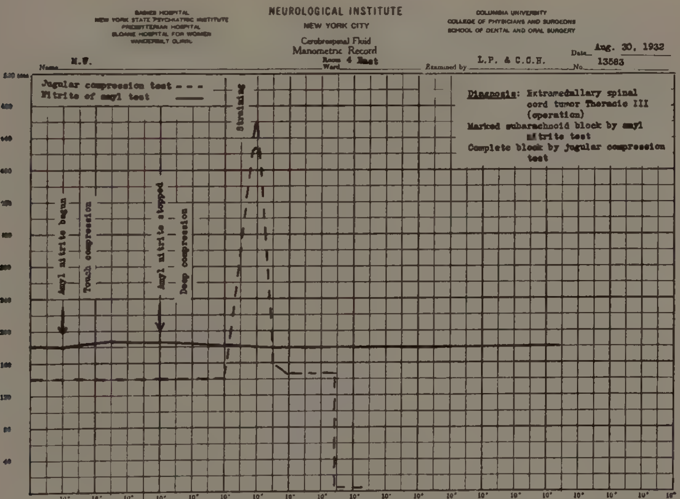
FIG. 8. Manometric chart showing a partial spinal subarachnoid block by the nitrite of amyl test and the absence of evidence of a block by the jugular compression test in a case of varicose veins of the spinal cord.



Name H.B.
Ward 3 West
Examined by H.M. & C.C.H.
Date May 14, 1932
No. 12401

Diagnosis: Spinal cord tumor (unverified)
Complete subarachnoid block by amyl nitrite test
Complete block by jugular compression test

FIG. 9. Manometric chart showing a complete subarachnoid block by the nitrite of amyl test and a marked block by the jugular compression test in a patient with a spinal cord tumor.

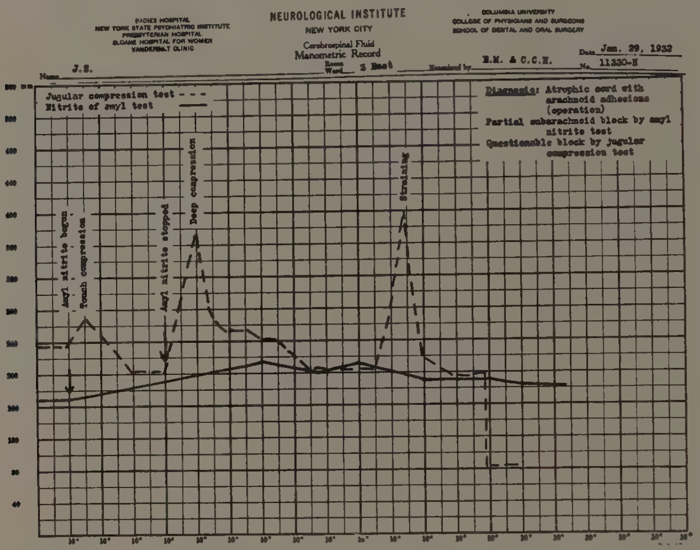


Name M.W.
Room 4 East
Examined by L.P. & C.C.H.
Date Aug. 30, 1932
No. 13583

Diagnosis: Extramedullary spinal cord tumor Thoracic III (operation)
Marked subarachnoid block by amyl nitrite test
Complete block by jugular compression test

FIG. 10. Manometric chart showing a marked spinal subarachnoid block by the jugular compression test and the nitrite of amyl test in a case of extramedullary spinal cord tumor.

est level; (d) the time required for the fall after deep compression has been discontinued; (e) the level to which the fluid falls; (f) the effects of straining; (g) the level to which the column of fluid falls after 7 cc. of spinal fluid have been allowed to escape (Figs. 7, 8, 9, 10, 11).


NEUROLOGICAL INSTITUTE
NEW YORK CITY
Cerebrospinal Fluid
Manometric Record

Name J. S.
Room / Ward 3 East
Examined by E.M. & C.C.E.
Date Jan. 29, 1932
No. 11330-E

Diagnosis: Atrophic cord with arachnoid adhesions (operation)
Partial subarachnoid block by amyl nitrite test
Questionable block by jugular compression test

FIG. 11. Manometric chart showing a partial spinal subarachnoid block by the nitrite of amyl test and a questionable block by the jugular compression test in a patient with localized adhesions of the arachnoid complicating meningomyelitis.

The main value of observations on the effect of straining, is to make it certain that the needle in the subarachnoid space is not obstructed. During the act of straining, there is always a rise of the column of fluid in the manometer. When there is a subarachnoid block, the rise during straining is due to the increase of pressure by the sudden congestion of the veins in the vertebral canal below the obstruction.

In some clinics, these complete tests are not carried out and apparently the value of careful observations of the time element is not appreciated. If the complete tests are not done, partial subarachnoid blocks may not be recognized, and this has, no doubt, led to the more frequent use of lipiodol.

In the majority of patients with tumors of the spinal cord, some degree of subarachnoid block can be demonstrated. In a few instances, however, the manometric tests fail to give positive evidence of compression of the spinal cord, and an obstruction in the spinal subarachnoid space is disclosed only at operation. In some patients in whom the symptoms and signs point to a lesion at a definite level of the spinal cord, the tests are inconclusive, and the examiner is not certain whether, upon compression of the jugular veins, a slight delay in the rise or fall of the fluid in the manometric tube has any significance.

The jugular compression test has certain disadvantages to which attention must be directed. The amount of pressure upon the jugular veins which is often necessary, may occasion the patient considerable discomfort. Furthermore, many individuals have a tendency to hold their breath and to strain during the period of jugular compression. Straining has a marked influence upon the pressure of the fluid (it produces a rise of the column of fluid in the manometer, whether or not there is a spinal subarachnoid block), and straining during the period of compression of the jugular veins may lead to errors of interpretation of the manometric curve. During the procedure the examiner must watch the patient closely, for the test can be properly done only when the patient is breathing normally and regularly.

In an individual with a stout neck, it may be difficult to make sufficient pressure upon the jugular veins, and in many patients, the carotid arteries may also be compressed somewhat. If the interruption of the current of blood in the jugular veins is not complete, the ensuing venous congestion within the cranial cavity will not be as marked, the expulsion of cerebrospinal fluid will not be as great, and therefore the level of the fluid in the manometer will not rise as high. If the carotid arteries are subjected to pressure at the same time as the jugular veins, the amount of blood in the cranial cavity during the manipulations may be less, the venous congestion from the compression of the jugular veins may not be as great, and again the pressure of the spinal fluid may not be raised as much as if the jugular veins alone had been compressed.

While these differences are of little significance in the presence of a complete subarachnoid block, they may interfere with the positive results of the test in the cases in which there is a partial obstruction in the spinal fluid pathway, and may suffice to explain many of the "questionable" blocks which have been observed.

The *nitrite of amyl test* is done in the following manner:

The nitrite of amyl is used in the form of glass pearls each containing 5 minims of the drug. The pearl of amyl nitrite is broken by the examiner and passed from side to side under the patient's nose for exactly thirty seconds. The time when the inhalation was begun and when it was discontinued is recorded. Manometric readings are taken every five seconds during the period of thirty seconds that the patient is inhaling the drug, and thereafter until the pressure has fallen to the level which had been recorded before the drug had been given. The time required for the pressure readings is three to four minutes.

In making the test, several precautions are necessary. The pearls made by some manufacturers are defective and the drug evaporates. In order to be certain that the broken pearl contains nitrite of amyl, it is only necessary for the examiner himself to recognize the well-known odor of the drug. While the amyl nitrite is being inhaled by the patient, the examiner should keep his finger on the patient's pulse, and the fact that the pulse has the characteristic bounding quality and that the face of the patient becomes flushed, is additional evidence that the physiological effects of the drug are being produced. Theoretically at least, there may be individuals whose nasal passages are so completely obstructed that the drug cannot reach and be absorbed by the nasal mucous membrane. Aside from the rarity of such a condition, the examiner who is feeling the pulse and observing the face of the patient, will quickly appreciate that the effects of the drug are not being produced.

Before the inhalation of the drug is begun, the patient is told that the substance he is going to inhale has a rather pleasant odor, that it causes no disagreeable sensation excepting a sense of fullness in the head, and that he should not cough but should breathe quietly.

After these facts have been explained, the patient usually breathes quietly and regularly during the period of inhalation of the drug.

1. The inhalation of the nitrite of amyl should be begun during the inspiratory phase of respiration.
2. The amyl nitrite should be inhaled for exactly thirty seconds.
3. The time when the inhalation of the drug is begun and when it is discontinued, should be recorded.
4. The pressure should be recorded every five seconds for a period of about three minutes beginning with the time the inhalation started.
5. The test should not be repeated for at least one hour.

When there is a block in the subarachnoid space, the jugular compression tests will show:

1. A complete block, when there is no rise of the fluid on both touch and deep compression, and when after removal of 7 cc. of spinal fluid, the fluid does not fall to a new level.
2. A partial block, when there is (a) no rise on touch compression but a diminished and slower rise and fall of the fluid level on deep compression, with perhaps very little fall in the manometric level when 7 cc. of fluid has been removed; or (b) very slight rise on touch compression and little and slow rise and fall of the level on deep compression.

When there is a block by the amyl nitrite test, the obstruction is

1. Complete, when there is a total rise of the column of fluid of less than 100 mm., and a rise of the column of fluid during the period of inhalation of less than 20 mm. and a delay in the beginnings of the rise of more than fifteen seconds (Fig. 9).
2. Marked, when the total rise of fluid is less than 100 mm. and a rise of the column of fluid *during* the period of inhalation of less than 20 mm., *or* a delay in the beginning of the rise of more than fifteen seconds (Fig. 10).
3. Partial, (a) a total rise of the column of fluid of less than 100 mm. and a rise of the level *during* the period of inhalation of the drug of between 20 and 35 mm.; (b) a rise of between 100 and

105 mm., and a total rise *during* the period of inhalation of not more than 20 mm. and a delay in the beginning of the rise of at least ten seconds (Fig. 8).

An obstruction to the free flow of fluid in the subarachnoid space can be demonstrated by these tests in tumors of the spinal cord and membranes, fractures and dislocations of the vertebrae with narrowing or distortion of the spinal canal, in adhesive processes between the membranes, and in some cases of varicosities of the spinal blood vessels. A spinal subarachnoid block is rare in kyphoscoliosis, even when the curvature is very marked.

The frequency with which subarachnoid block is observed will be considered in the chapters devoted to diseases and to injuries of the spinal cord.

Compression of the veins of the neck is also a procedure of value during operation for spinal cord tumor or other spinal lesion, if the growth has not been found in the area of the cord that has been exposed. This procedure was suggested by Mixter. If the growth is above the level exposed, then compression on the veins of the neck will not be followed by a flow of fluid from higher up. The procedure is also useful when the surgeon desires to make certain that the subarachnoid space is patent above the level at which he has been operating (see laminectomy for spinal cord tumor and for other lesions).

DOUBLE SPINAL PUNCTURE AND ITS VALUE

As soon as it became known that in spinal compression the fluid obtained by lumbar puncture showed certain characteristic changes, the idea occurred to a number of investigators to study the fluid obtained by puncture of the subarachnoid space above the location of the compression and to compare it with the fluid obtained by lumbar puncture.

Marie, Foix and Robert (1913), were among the first to investigate this subject. Careful investigations were made by Ayer and his co-workers. Ayer (1920, 1921), worked out a method of performing puncture of the cisterna magna, a procedure which, in his hands at least, has been without danger. By this method of cistern puncture, Ayer obtained fluid which was always above the site of a spinal compression, and he compared the fluid thus obtained with the fluid withdrawn by lumbar puncture. In cord tumors, the difference between the two fluids is so marked that Ayer has recommended this double puncture as a valuable procedure for the diagnosis of spinal block.

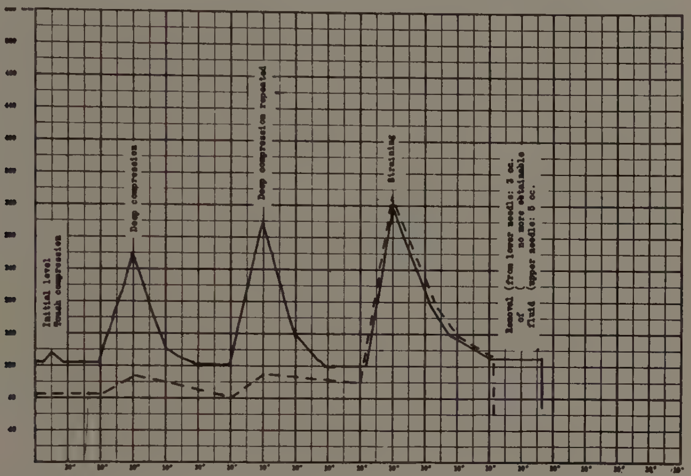


FIG. 12. Manometric pressures in the case of a chondroma at the level of the second lumbar vertebra. The solid line indicates the result of lumbar puncture below the first lumbar vertebra; the broken line the result from lumbar puncture below the fourth lumbar vertebra. The readings were made simultaneously.

Ayer (1921) made valuable manometric studies of the pressure of the cerebrospinal fluid above the level of a spinal block (by the cistern puncture) as compared with the pressure of the fluid below the level of the obstruction (by the lumbar puncture). If there is no block in the spinal subarachnoid space, the fluid in both manometers will rise to about an equal height upon deep expiration and upon pressure upon the veins of the neck. If, on the other hand, there is a subarachnoid block, the fluid in the upper manometer will rise as in the normal individual, while the fluid in the lower manometer will either not rise at all or will rise relatively little and more slowly after compression of the veins of the neck. Ayer deserves much credit for the careful work he has done, but it must be remembered that cistern puncture is a procedure not without danger, and I know of several instances in which, even with experienced surgeons, bleeding into the fourth ventricle occurred. In the large majority of instances, lumbar puncture with manometric tests and examination of the fluid will give all necessary information.

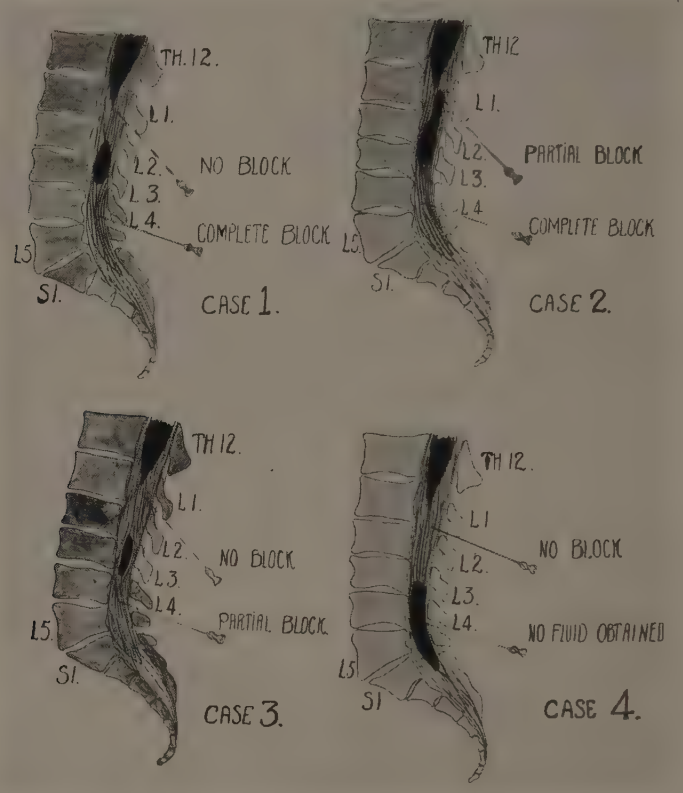


Fig. 13. Double lumbar punctures in tumors of the conus and the cauda equina. (See also Fig. 14.)

In tumors of the conus and cauda equina, double lumbar punctures are of value for localization. The lumbar puncture needles may be introduced at one sitting and the manometric tests and removal of fluid for the laboratory examination may be removed synchronously, or the second needle may be introduced into the subarachnoid space after the tests with the first needle have been completed. Elsberg and Cramer (1930) showed that the information gained may be the following (Figs. 12, 13, 14):

(a) The typical case in which a complete subarachnoid block is demonstrated by puncture through one of the lower lumbar interspaces, and no block at the first (or second) interspace. This signifies that the tumor is situated somewhere between the first (or second) and the fourth (or fifth) lumbar vertebra (Fig. 13, case 1).

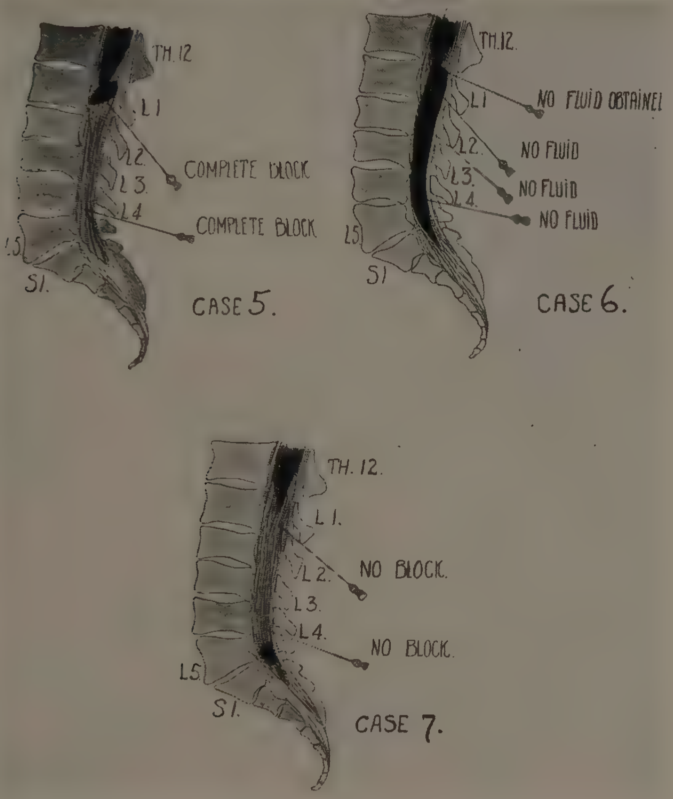


FIG. 14. Double lumbar punctures in tumors of the conus and cauda equina. (See also Fig. 13.)

(b) There is a complete subarachnoid block at the level of the fourth lumbar vertebra and a partial block at the first. This means that the tumor extends somewhat above the first lumbar vertebra but lies mainly somewhere between the first and the fourth lumbar vertebra (Fig. 13, case 2).

(c) There is a partial block at the fourth lumbar vertebra and no manometric evidence of block at the first. This means that the tumor lies somewhere between those two levels but is not of sufficient size to obliterate completely the subarachnoid space above the lower level (Fig. 13, case 3).

(d) No fluid obtainable at the fourth lumbar, and fluid easily withdrawn and no subarachnoid block at the first. This means that the lesion is below the level of L 1 and that it is probably of large size (Fig. 13, case 4).

(e) Complete subarachnoid block at the fourth *and* at the first lumbar vertebrae. This indicates that the tumor lies near the tip of the conus (Fig. 14, case 5).

(f) No fluid obtained by puncture through any of the lumbar interspaces. This indicates that there is a large tumor which fills up the lower part of the vertebral canal, and which may extend upward above the conus (Fig. 14, case 6).

(g) No block at the fifth and no block at the first lumbar interspace. This means that the tumor, if it is a tumor, is situated in the sacral part of the vertebral canal (Fig. 14, case 7).

ALTERATIONS IN THE CEREBROSPINAL FLUID IN SPINAL DISEASES

Changes in the Color of the Fluid. Normal cerebrospinal fluid is clear and colorless; but changes in color, especially more or less xanthochromia, occur in a variety of intracranial and spinal affections.* The fluid obtained by lumbar puncture is often yellow after intracranial diseases or injuries; in intracranial tumors in or near the ventricles; in many cases of subdural hematoma; after fractures or other trauma to the skull; after spontaneous subarachnoid hemorrhage; and in cerebrospinal meningitis.

Yellow spinal fluid may be found in a variety of spinal diseases: after old or recent fracture of the spine; in Pott's disease; in inflammatory diseases such as spinal meningitis, extradural abscess secondary to osteomyelitis of a vertebra, and the like; and in radiculitis of the roots of the cauda equina. In chronic progressive spinal diseases, the occurrence of xanthochromia always justifies the suspicion of compression of the cord by a new growth. The deepest yellow fluids are found in tumors and other lesions of the conus and roots of the cauda equina.

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* If the xanthochromia is slight, it may be difficult to recognize that the fluid has a yellow tinge, unless the test tube is compared to one which contains water. If one looks through the fluid in the axis of the test tube, slight changes in the color can often be appreciated.

The frequency of xanthochromia in 209 patients with spinal cord tumors of my series is shown in Table III:

TABLE III

FREQUENCY OF XANTHOCHROMIA IN 209 CASES OF CORD TUMOR

Situation	*Fluid*		*Totals*
	Clear	*Xanthochromic*	
Extramedullary	67	70	137
Extradural	37	18	55
Intramedullary	10	7	17
Totals	114	95	209

Xanthochromia is more frequent in tumors as well as in other lesions in the lower thoracic and lumbosacral regions, and occurs almost regularly in large tumors of the conus and cauda equina. Of the 209 cases of Table III the spinal fluid was yellow in 32 per cent of tumors of the cervical and upper thoracic, and in 76 per cent of tumors below the level of the seventh thoracic segment of the spinal cord.

Increase of protein and of globulin is frequent in diseases of the spinal cord and especially in cord compression by new growths. It is also not unusual to find a moderate increase in old fracture dislocations of the vertebrae. When tested by Ayer's method (sulphosalicylic acid test) the total protein in normal fluid may have as high a value as 40 mg. in 100 cc. of fluid, but values as high as 50 mg. are found so often that this must be considered a high normal. It is not uncommon to find moderate increase of total protein (up to 70 mg. per 100 cc.) without any other definite evidence of a lesion in the cranial cavity or spinal canal, and for the present, such cases remain unexplained. An increase of protein (up to 100 mg.)

is not rare in diseases of the spinal cord, such as multiple sclerosis and syringomyelia; and high protein values are usual in spinal syphilis and in inflammatory diseases such as meningitis and spinal extradural abscess.

In tumors of the spinal cord, the total protein may be as high as 2000 mg. to 100 cc. or more. In general, the protein values are less than 200 cc. in most extradural growths, they are apt to be less in the meningeal than in the perineurial fibroblastomas, and they are often very high in intramedullary tumors. The highest protein values are found in growths of the conus and cauda equina, and in acute inflammatory processes.

It happens, frequently, that the fluid obtained by lumbar puncture is blood-stained or actually contains much blood. When the fluid is bloody, a corrected total protein content can be obtained by subtracting 4 mg. per 100 cc. of fluid for every 5000 red cells (Solomon, Dailey and Fremont-Smith). According to the same authors, corrected white cell counts can be obtained by subtracting one white cell for every 500 red cells in the fluid.

Spontaneous coagulation of the fluid that has been withdrawn, often occurs in acute and chronic inflammatory processes in or around the spinal cord or membranes, after a recent cranial or spinal trauma with blood in the cerebrospinal fluid, and especially in tumors. Spontaneous coagulation always means a protein excess, but is not always proportional to the amount of protein. Fluids with very high total protein may not coagulate spontaneously. In tumors of the spinal cord, spontaneous clotting is most frequent in the xanthochromic fluids which occur with tumors of the conus and cauda equina.

While the total protein can be quantitatively determined by the sulphosalicylic test (and other similar tests), a satisfactory method for the exact determination of the amount of globulin has not yet been devised.

The *cells* in normal cerebrospinal fluid vary from 0 to 5 in number, and are lymphocytes. The number of cells is increased in inflammatory processes within or outside of the membranes, in meningovascular lues and in many intramedullary growths. Occasionally, also, the cell count is larger than normal in extramedullary tumors of the spinal cord, especially in the large growths of the conus and cauda equina.

THE COMPRESSION SYNDROME

The combination of xanthrochromia, increase of total protein, spontaneous coagulation, and normal cell count, first described by Froin (1903), and called Froin's syndrome, has been, in my experience, characteristic only of the large tumors of the conus and cauda equina which I have called "giant" tumors.

The so-called syndrome of Nonne (1910)—excess of protein and globulin without increase of cells—is more often found in tumors at other levels. But a combination of the features of the fluids described by Nonne and one of the characters of the fluids first described by Froin—xanthochromia, normal cell count and increase of total protein—is often found in extramedullary and extradural tumors above the lower thoracic levels of the spinal cord. In intramedullary growths xanthochromia is rare, but a combination of increase of protein and pleocytosis is more frequent. Sicard and Foix (1912), claimed that a marked disproportion between the increase of protein and the number of cells—a "dissociation albumino-cytologique" as they called it—occurred more often in intradural than in extradural tumors. In a sense Sicard and Foix are correct, for the total protein is apt to be lower in extradural than in some varieties of intradural growth.

CHAPTER III

THE ROENTGEN RAY DIAGNOSIS OF DISEASES OF THE SPINAL CORD, MENINGES, AND VERTEBRAE

By Cornelius G. Dyke, M.D.

THIS SECTION IS DEVOTED TO THE ROENTGEN RAY EVIDENCE OF THE VARIOUS DISEASES WHICH AFFECT THE SPINAL CORD, THE meninges, and the extradural soft tissues and bone. The author has endeavored to present the characteristic diagnostic features of the various diseases whenever possible. In many instances the evidence in the roentgenograms may not be sufficiently definite to permit a differentiation of the tumor type, but in the vast majority of instances there should be no difficulty in differentiating neoplasms from infectious diseases, from traumatic processes or from congenital anomalies.

A considerable number of illustrations depicting the characteristic changes produced by the various diseases has been presented, so that the reader may better visualize the pathologic disturbances. It is impossible, of course, to illustrate all types of the various lesions that have been described in the text. The tumors have been more fully illustrated than any of the other lesions, because of their great importance in spinal cord surgery.

The examination of the spinal canal by means of contrast media has been described. The use of lipiodol in the detection of posterior herniation of the nucleus pulposus and of hypertrophy of the ligamentum flavum has been described separately rather than under the heading of lipiodolography.

SKIN MARKING AS AN AID FOR SURGICAL PROCEDURES

After the lesion has been localized, it is well to indicate on the skin the site of the pathologic process in order that the surgeon may be able to operate at the correct segmental level. At the Neurological Institute, it has been the practice for a number of years to fluoroscope the patient, and during this procedure to indicate with a pair of scissors or hemostat, or some other radio-opaque instrument, the position of the pathologic process. Following this, a small amount of methylin-blue or indigo-carmine is injected into the

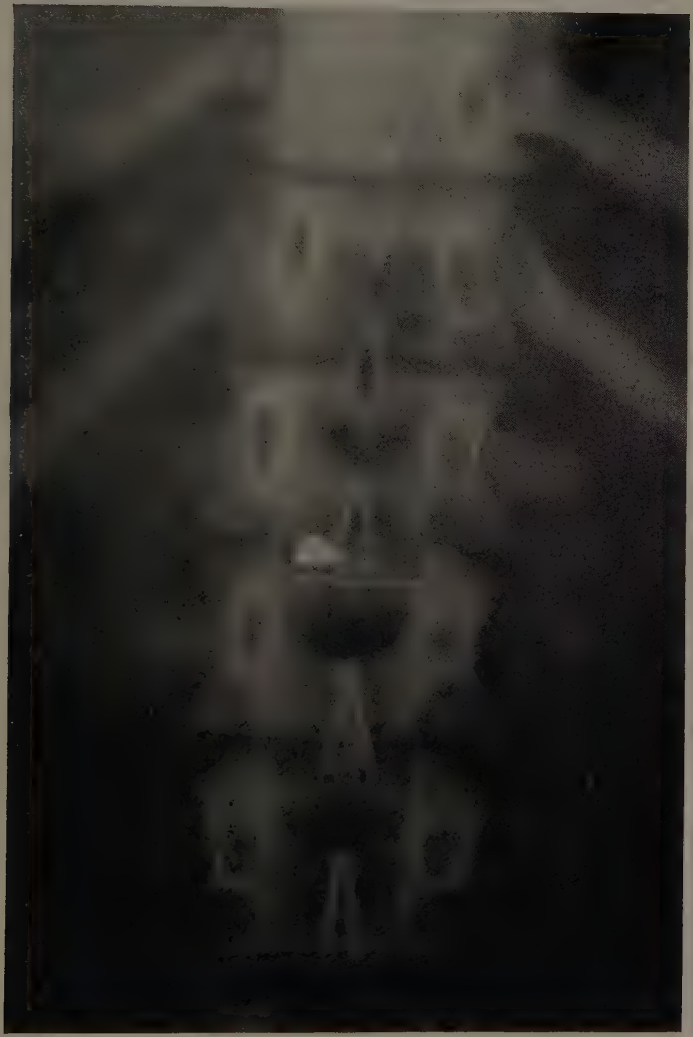

FIG. 15. R.R.* No. 31431. Lead arrow indicating site of intradermal injection of dye for the purpose of identifying level of the lesion at time of operation.

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* R.R. in this and following legends is used as an abbreviation for Roentgen Ray.

skin slightly lateral to the midline. A lead arrow is then placed over the dye (Fig. 15). Anteroposterior stereoscopic roentgenograms are made, and the position of the dye as indicated by the lead arrow is checked. This is indicated in the report and the surgeon then knows the position of the dye in relation to the vertebrae. The dye will show after the skin has been prepared with iodine and alcohol immediately prior to the surgical incision. This enables the surgeon to perform the laminectomy exactly at the site desired. In some cases the fluoroscopy is omitted and the dye is injected intradermally, a lead letter placed on the discolored area, and anteroposterior roentgenograms are made. The film is then developed and the position of the lead letter or arrow is noted in relation to the vertebrae.

CONGENITAL ANOMALIES

Bone

Congenital anomalies of the spine are common, particularly at the ends of the spinal column. Frequently a gap is noted in the posterior portion of the arch of the first cervical vertebra, which is of no clinical significance. Likewise, there are other types of congenital anomalies of the cervical vertebrae, such as fusion of the bodies, hemivertebrae, and, in the lumbar region, failure of two laminae to join with the center of ossification for the spinous process. In the lumbar portion of the canal there frequently are found anomalies in the articular facets, particularly between L 5 and S 1, or L 4 and L 5.

It is not within the province of this chapter to discuss all the types of congenital anomalies which may occur within the spinal column but only those having significance from a neurosurgical point of view.

Congenital Synostosis of the Cervical or Cervicothoracic Spine (Klippel-Feil Syndrome). Willett and Walsham (1880), reported in a paper entitled "An Account of the Dissection of the Parts Removed after Death from the Body of a Woman Subject of Congenital Malformation of the Spinal Column, Bony Thorax and Left

Scapular Arch, with Remarks on the Probable Nature of the Defects in Development Producing the Deformities," the first case of what has since become known as the Klippel-Feil syndrome. The deformity of the spinal column in this case consisted of a loss of its posterior convexity; a sharp lateral curve with the convexity to the left in the upper dorsal region, corresponding to the absence of the right half of the third dorsal vertebra; a compensatory curve in the lower dorsal region where four and one-half dorsal vertebrae were missing. With these spinal changes there were associated marked deformities of the sternum and ribs; in fact, five ribs were missing on the right, and four on the left side of the chest.

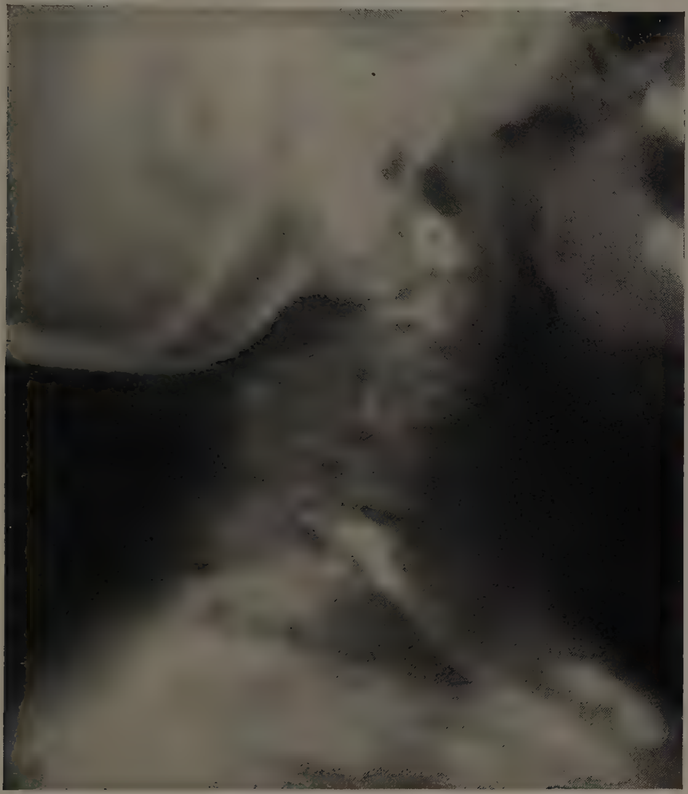

FIG. 16. R.R. No. 45413. Fusion and malformation of cervical vertebrae in a case of Klippel-Feil syndrome.

Hutchinson (1894) exhibited, before the Pathological Society of

London, a specimen obtained from a child in whom the left shoulder was fixed high up in the neck under the occiput. The laminae of the third, fourth, fifth, and sixth cervical vertebrae did not meet in the midline and were fused on the left side. From the fused mass there proceeded outward a plate of bone terminating in a process of cartilage with which the scapula articulated by its upper and inner angle.

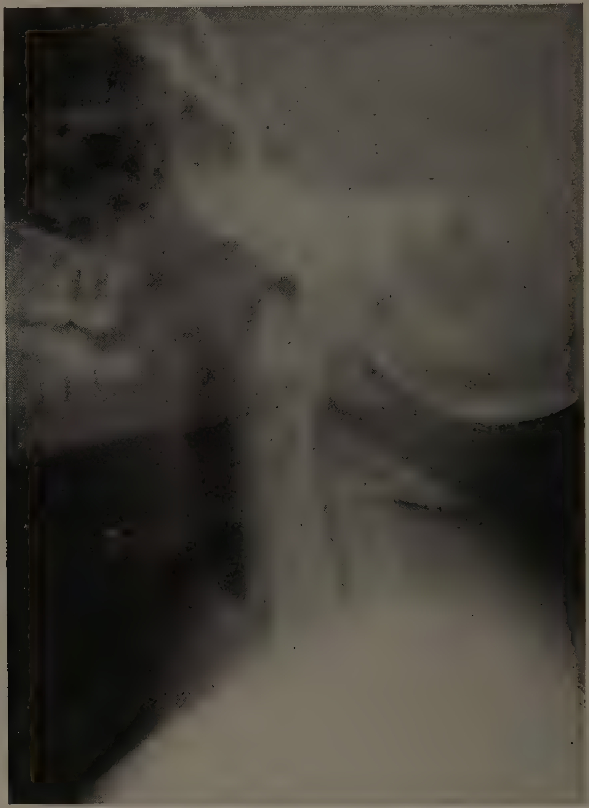

FIG. 17. R.R. No. 43389. Deformity and fusion of cervical vertebrae in a case of Klippel-Feil syndrome.

Clarke (1906) reported that from birth a boy, aged four years, had his head fixed so that the chin was close to the sternum and there was no movement in the cervical spine. Clarke was the first to make skiagrams of the spine, which showed extensive abnormalities in the upper dorsal and cervical regions, and the presence of bilateral

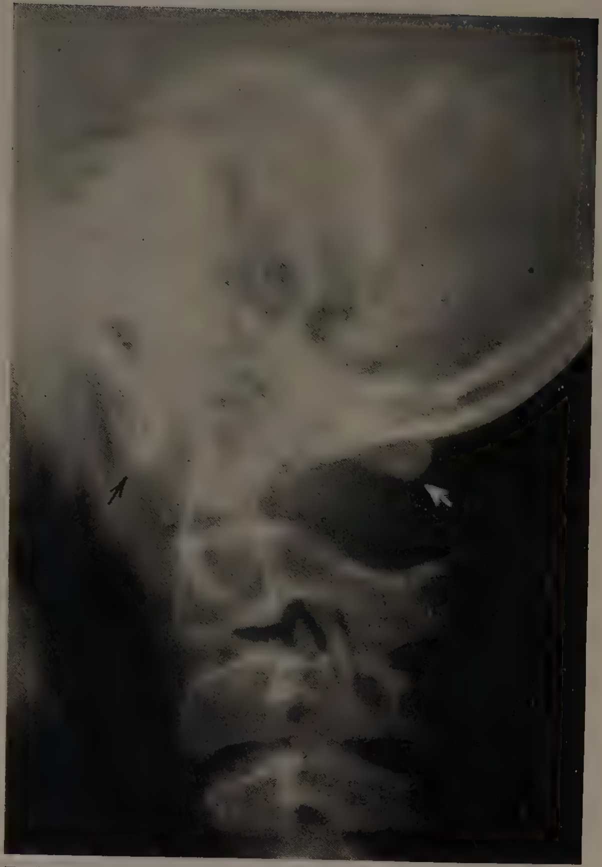

Fig. 18. R.R. No. 31231. Fusion of atlas and occipital bone (arrows).

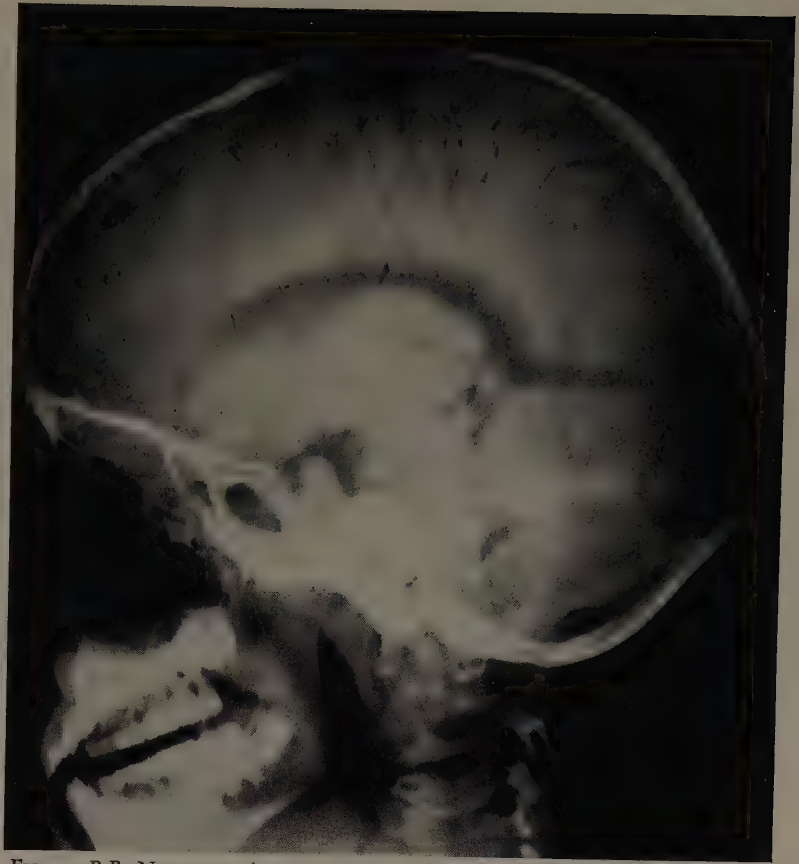

Fig. 19. R.R. No. 31231 (same case as Fig. 18). Encephalogram showing normal lateral, third, and fourth ventricles, basal cisterns, and cerebral sulci.

cervical ribs. Clarke operated upon the patient and followed this by massage. This treatment gave the boy a short neck with all the natural movements. The details of the operative procedures are unknown.

Klippel and Feil (1912) reported in detail the case of a forty-six-year old male tailor who was found at necropsy to have fused cervical vertebrae and a posterior spina bifida occulta. The fused mass of cervical spine did not contain a recognizable axis or atlas. It probably contained some dorsal vertebrae since there were four pairs of ribs and only eight normal dorsal vertebrae distal to the mass. They expressed the view at this time that the etiology was intra-uterine inflammation or trauma. Since the recording of these cases, many others have been reported and numerous excellent articles have appeared.

The Klippel-Feil syndrome is dependent upon a synostosis and malformation of a number of the cervical, or cervical and dorsal vertebrae. The individual bodies of many of the vertebrae frequently cannot be identified (Fig. 16). Narrowing of one or more vertebral bodies, either anteriorly or at the lateral margins, is common. In addition to these deformities, there is usually imperfect closure of the dorsal portion of the spinal arches (spina bifida occulta). Marked deformity, fusion or absence of the articular facets and processes of one or more vertebrae commonly is seen (Fig. 17). Kyphosis, scoliosis or kyphoscoliosis of the cervical spine also are common. The changes noted above are not limited to the cervical vertebrae, for in some instances striking deformities of the dorsal spine occur. The process is undoubtedly due to an arrest in the development of the involved vertebrae. The abnormalities of the spine at times are associated with changes in the ribs or in the scapulae as described above.

Congenital Malformations of the Atlas. Anomalies of the atlas are common. The most frequent is spina bifida occulta and this has no clinical significance in the vast majority of cases.

In rare instances the atlas may fuse with the occipital bone. The fusion may take place between the arch and the squamous portion of the occipital bone, between the anticular facets of the atlas and the condylar processes of the occipital bone, or at both these points

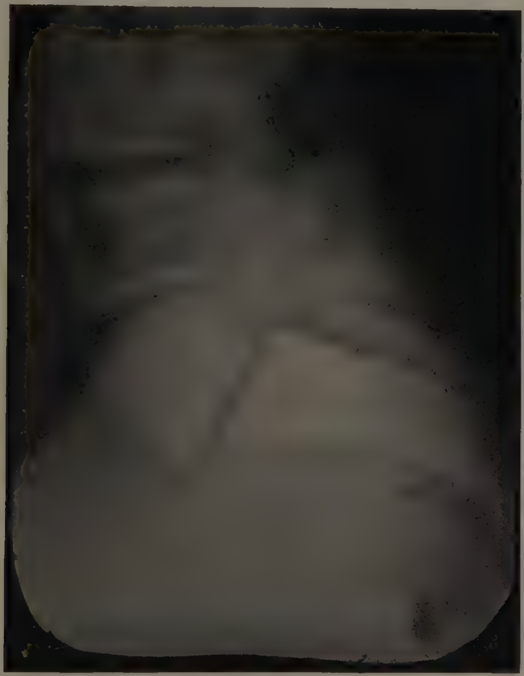

FIG. 20. R.R. No. 24423. Spondylolisthesis of fifth lumbar vertebra. (Retouched.)

(Figs. 18, 19). The deformities of the atlas are frequently associated with others involving the occipital bone, such as platybasia and narrowing of the foramen magnum. These conditions, however, will not be discussed here since they are not spinal lesions.

Spondylolisthesis. Spondylolisthesis is the forward displacement of the lumbar vertebrae with consequent contraction of the pelvis. The condition is best recognized in the roentgenogram in the lateral view. In this position, whether the view was made with the patient erect or horizontal, the body of the fifth lumbar vertebra is ventral to the first sacral segment. The amount of displacement varies. With the anterior displacement of the fifth lumbar body, the other vertebrae cephalad to it are also displaced anterior to their normal position. The fourth lumbar vertebra is occasionally affected. In many of the cases there is a fibrous defect in the arch at the junction of the laminae and the lateral masses. Because of this, the body and pedicles of a vertebral segment may move forward, but the spinous process, laminae and the articular processes may remain in their normal position or even be displaced dorsally (Fig. 20).

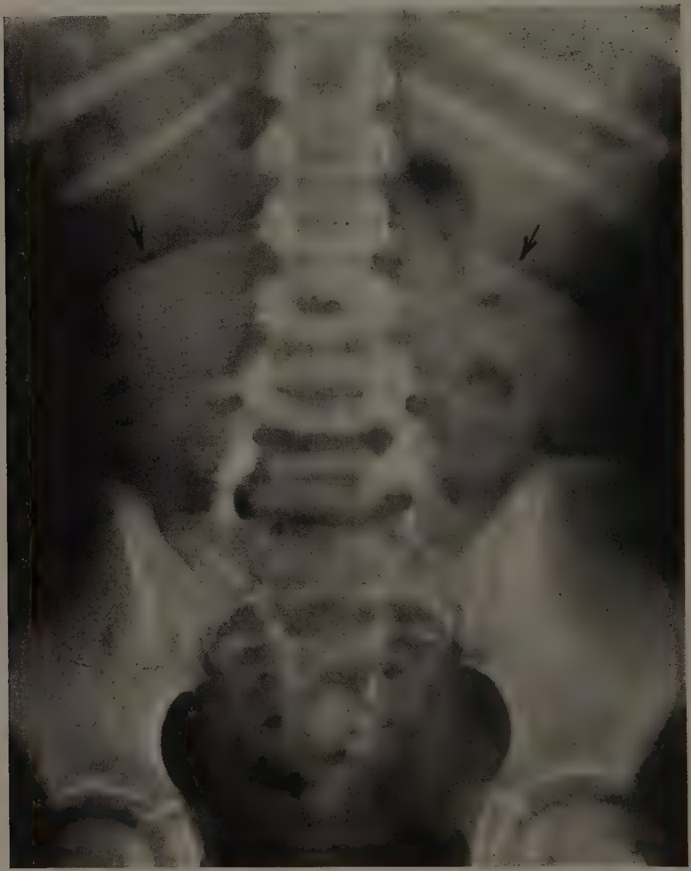

Fig. 21. R.R. No. 22781. Anteroposterior view showing defect in the arches of the lumbar vertebrae in a case of spina bifida and the associated meningocele (arrows).

Meninges, Brain and Spinal Cord

Arnold-Chiari Syndrome. Arnold-Chiari malformation consists of a caudal elongation and extension of the cerebellum and brain stem into the cervical portion of the spinal canal, and is associated with spina bifida.

The roentgenograms in the majority of cases show an enlargement of the head due to internal hydrocephalus. In addition, there is present a spina bifida in the lumbar or dorsolumbar region.

Meningocele or Meningomyelocele. In meningocele or meningomyelocele there is a defect in the dorsal portion of the arches of one or more vertebral segments. In most instances the laminae are equally affected on the two sides, while in other cases, maldevelopment is considerably greater on one side than on the other. In addition to the defect in the dorsal portion of the arch or arches, the pedicles may be considerably separated so that the interpediculate distance is increased (Fig. 21). In some cases the pedicles are flattened, particularly in the older patients. In addition to the changes noted in the bone in the anteroposterior roentgenogram, a variable-sized soft tissue mass can be seen opposite the vertebrae that have developmental defects. In the lateral roentgenograms, particularly in the films that are made to show the soft tissue detail, the meningocele may be clearly outlined (Fig. 22). Meningoceles do not contain calcium.

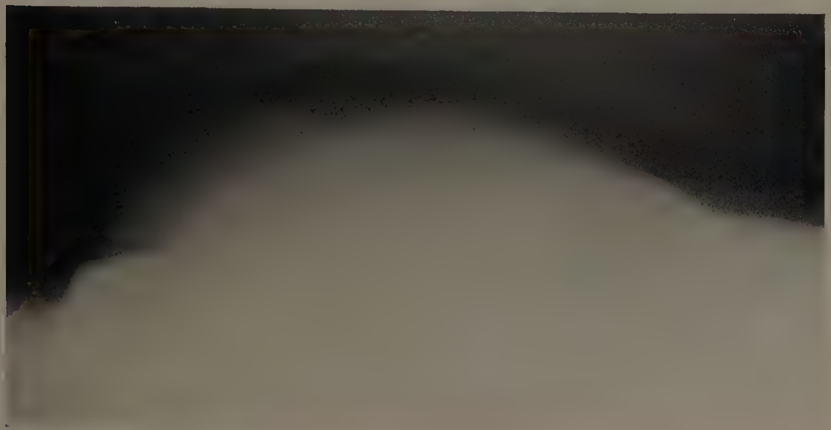

FIG. 22. R.R. No. 22781 (same case as Fig. 21). Lateral view showing the soft tissue mass formed by the meningocele.

TRAUMATIC CONDITIONS OF THE SPINE

FRACTURE OF THE VERTEBRA

Ordinary Type. Fractures of the spine are usually produced by hyperflexion of the body, and most of the compression fractures

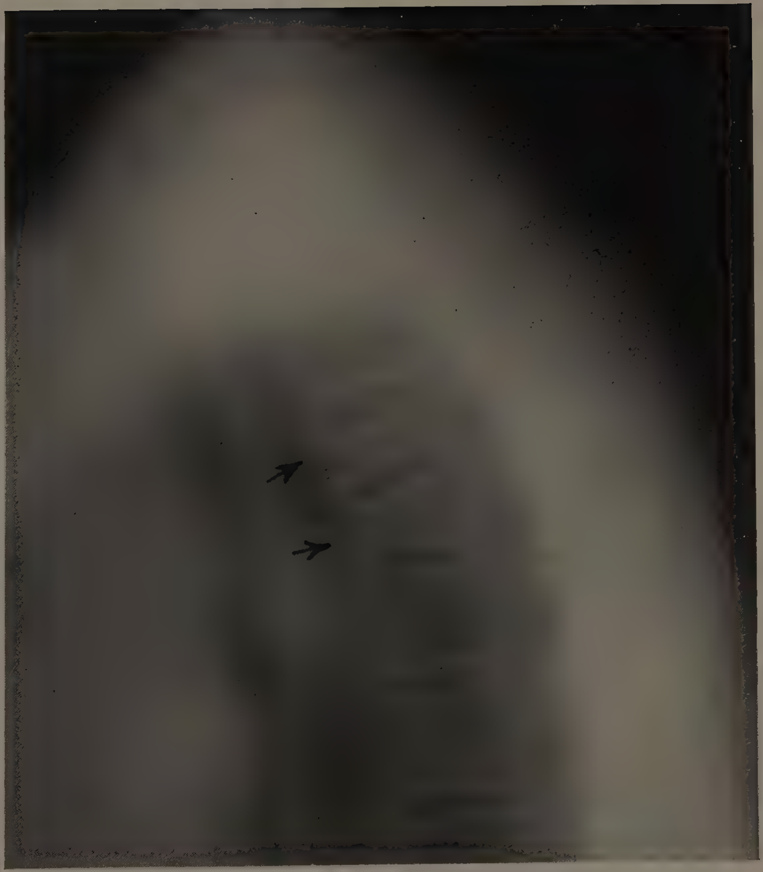

FIG. 23. R.R. No. 46722. Laminagram showing compression fractures of the bodies of T 3 and T 4 (arrows).

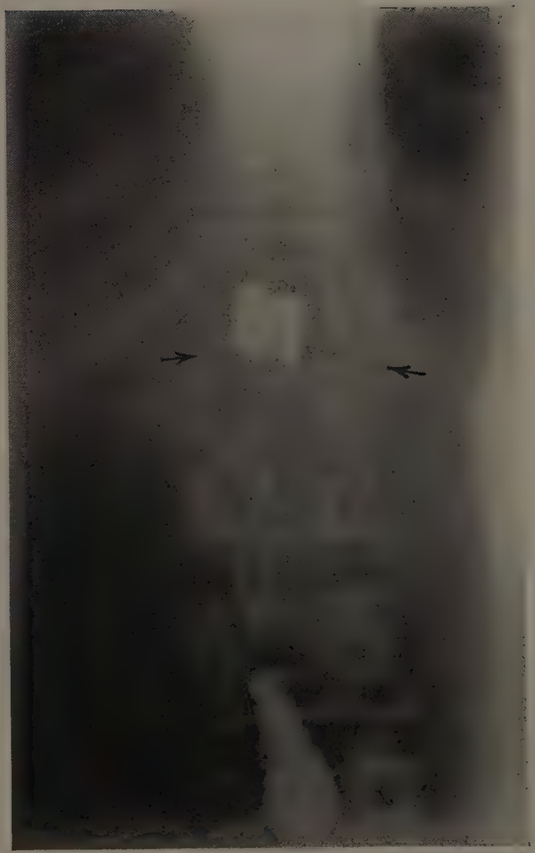

FIG. 24. R.R. No. 45817. Old compression fracture of the body of L 1 with encroachment on the spinal canal as indicated by the obstruction to the lipiodol (arrows).

occur in the dorsolumbar region, that is, T 12, L 1 and L 2; or in the cervical region, particularly in the three lower vertebrae. Extreme flexion of the body is the usual force which produces a compression fracture of a vertebral body. This portion of the vertebra as a result is usually wedge-shaped; that is, the ventral margin of the body is considerably narrower than the dorsal portion (Fig. 23). Not infrequently, irregular fragments are separated from the vertebra. In some of these cases the posterior portion of the body of a vertebra may project into the spinal canal and thereby compromise its capacity (Fig. 24). It is uncommon to have narrowing of an intervertebral disc adjacent to a fractured vertebral body. Occasionally, one lateral margin of the vertebral body will be more compressed than the other. Fracture of the arches of the vertebrae are infrequent and are usually due to penetrating wounds. Fractures of the spinous processes are either due to penetrating instruments or muscle pull.

Old compression fractures of the body of a vertebra may show no additional changes to those described for a recent one. However, in many instances after a considerable period of time, and this varies in the individual patient, hypertrophic spurs of varying size appear at the margins of the fractured vertebra.

Pathological Fracture of the Vertebra. Pathological fracture of a vertebral body is usually secondary to tumor, often metastatic from a carcinoma of the breast. Infrequently, it is due to a primary tumor of the vertebral body, such as a giant-cell sarcoma or myeloma. In that rare condition known as "Cushing's syndrome," and in hyperparathyroidism, advanced osteomalacia with multiple compressions of the bodies of the vertebrae frequently occurs, particularly in the dorsal region (Fig. 25).

Scheuermann's Disease (Kyphosis Juvenilis). Kyphosis juvenilis involves particularly T 10 and T 11, and may involve other vertebrae especially in the mid-dorsal region. Each vertebral body is somewhat wedge-shaped and usually several vertebrae are involved (Fig. 26), but the process may be limited to a single vertebral segment. The intervertebral discs between the involved vertebrae are quite irregular in outline, and this is also true of the margins of the vertebral bodies. The epiphyses are often larger anteriorly than posteriorly, and are irregular in outline. This condition, at the present time, is considered due to an interference with the blood supply to the epiphysis or epiphyses of the bodies. The cause, therefore, is similar to that in Legg-Perthe's disease of the hip, and Osgood-Schlatter's disease of the tibial tubercle. It is common for kyphosis juvenilis to be present in young patients with spinal cord tumors. The type of tumor does not appear to affect the frequency of its occurrence. This fact also suggests that it is pressure upon the blood supply of

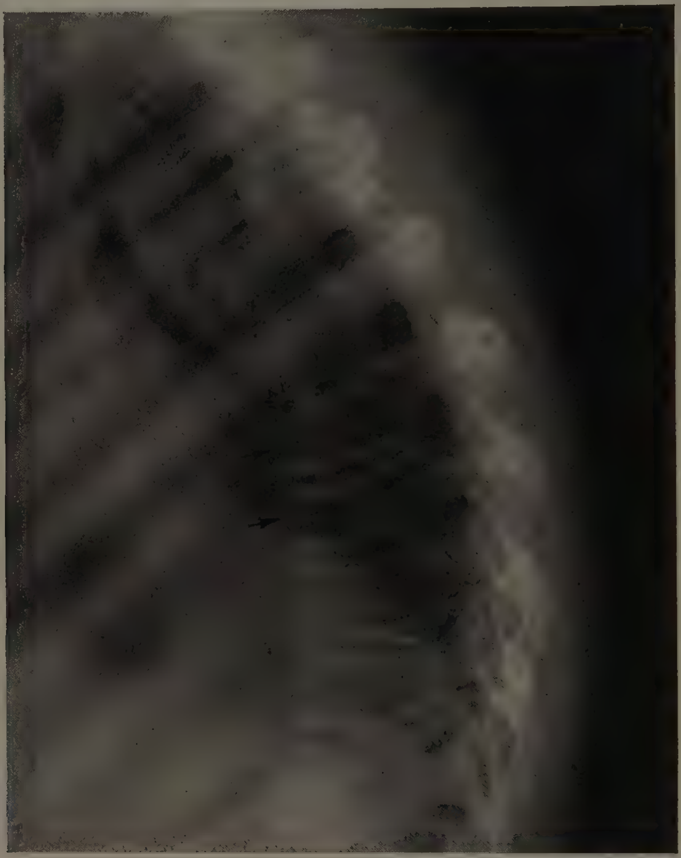

FIG. 25. R.R. No. 44911. Compression fractures of the bodies of a number of the mid-dorsal vertebrae in a patient with pituitary basophilism.

the epiphyses that is the important factor in the production of the disease.

Kümmel's Disease. Kümmel's disease, first described in 1891 by the man whose name it bears, is the term applied to a condition in which trauma has occurred to the spine, but shortly thereafter a roentgen examination of the spine reveals it to be normal. Subsequent roentgenograms, however, show a compression of the body of a single vertebra. Rarefaction of the body of a vertebra precedes the compression. In fact, only rarefaction of the body may take place. At the present time it is believed that the condition is due to an

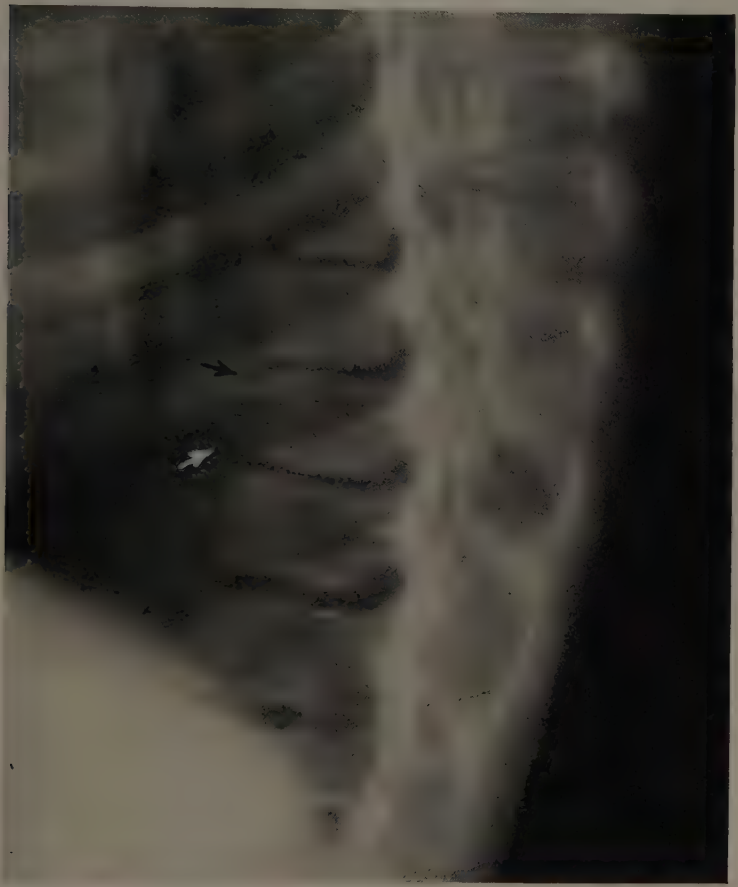

FIG. 26. R.R. No. 25207. Scheuermann's disease (kyphosis juvenilis) showing the irregularity of the margins of the body of T 9 (arrows).

interference with the proper nutrition of the vertebral body which produces atrophy and if this process is carried sufficiently far, it results in compression of the body. As Sussman and Kugel point out, Kümmel's disease, therefore, is not truly a fracture of the vertebra due to injury but it is dependent upon secondary nutritional changes occurring in the body.

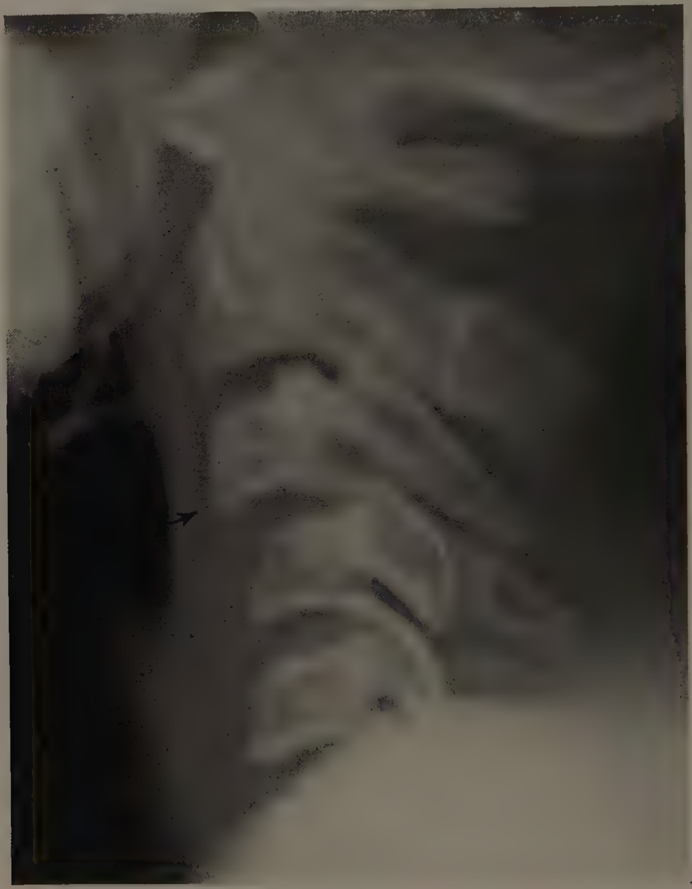

FIG. 27. R.R. No. 37451. Dislocation of C 3 on C 4 (arrows).

DISLOCATION OF THE VERTEBRA

Dislocation of one vertebra upon another occurs frequently in the cervical (Fig. 27) and in the dorsolumbar regions. The relation of the vertebrae is changed usually in the anteroposterior plane and less often in the lateral plane. In the vast majority of instances the articular facets are still in contact with each other at their surfaces, and only a moderate displacement occurs at these points. Even with the articular surfaces in contact, a fairly marked kyphosis often exists. In the case of an anterior dislocation, the dorsal margin of the body of the adjacent vertebra distal to the dislocated one encroaches upon the spinal canal.

Dislocations of the atlas are usually associated with fracture of the dens, and the direction of displacement is ventral. Dislocations of the atlas or axis are infrequent.

Unilateral dislocation of a vertebra in which the inferior articular facet occupies an anterior position to the next distal facet is difficult to visualize in the roentgenogram and calls for the utmost precision in obtaining stereoscopic films in the lateral, oblique, and anteroposterior positions. It must constantly be borne in mind that in the cervical region there is normally considerable motion between the various vertebrae. A well founded clinical diagnosis of unilateral subluxation or dislocation of an articular facet and process, usually is more reliable than a roentgenological diagnosis. The roentgen diagnosis in most instances is supportive to the clinical diagnosis.

Posterior Herniation of the Nucleus Pulposus

Roentgen Evidence

Stereoscopic Roentgenograms

The most significant evidence in the roentgenogram of the spine which permits a suggestive diagnosis of herniation of the nucleus pulposus is *narrowing of an intervertebral disc* (Fig. 28). In most of the cases the entire disc is decreased in width. However, in not a few cases, there is narrowing of only one side of the intervertebral disc (Fig. 29) or of the posterior margin alone. The thinning of the intervertebral disc may be as little as 2 or 3 mm., but often it amounts to as much as 5 mm.

It is of the greatest importance in estimating the thickness of the intervertebral disc to have films made so that the primary beam of roentgen rays is directed through the disc. In this connection it should be stated that inasmuch as most herniations of the nucleus pulposus occur in the lower lumbar region, it is necessary for the interpreter of the roentgenograms to know the normal thicknesses of the various intervertebral discs. A good rule to follow in this regard is, that from the lower dorsal region, about T 9 or T 10, the intervertebral discs progressively increase in thickness until the fifth lumbar vertebra is reached. Some of the intervertebral discs between the above-mentioned points may be the same thickness between two adjacent segments. However, the disc caudal to the one just above it should be the same size or slightly larger than the one above it, and when the caudal disc is narrower than the one just cephalic to it,

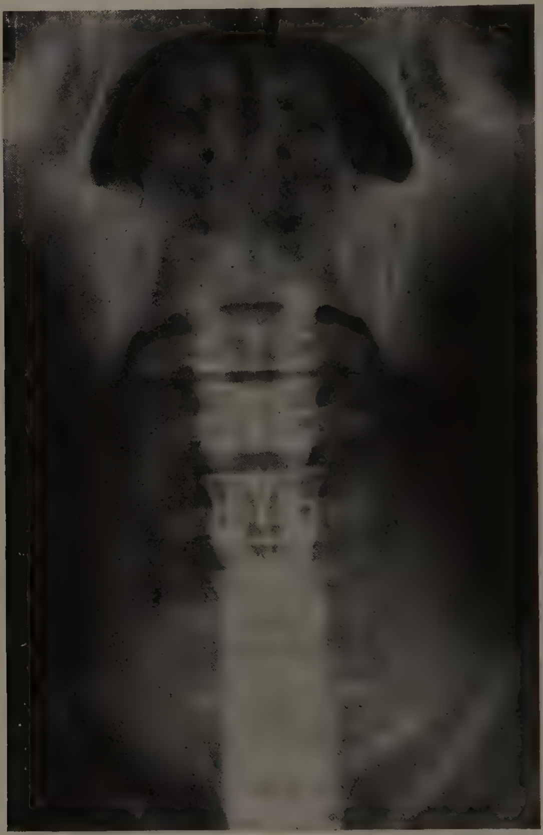

FIG. 28. R.R. No. 30965. Roentgenogram shows narrowing of entire intervertebral disc (arrows) between L 4 and L 5, due to posterior herniation of the nucleus pulposus.

the intervertebral cartilage is abnormally thin. The intervertebral cartilage between L 5 vertebra and S 1 vertebra may be of any thickness and of any shape and still be normal. Therefore, its appearance should not be used in the establishment of a diagnosis of posterior herniation of the nucleus pulposus.

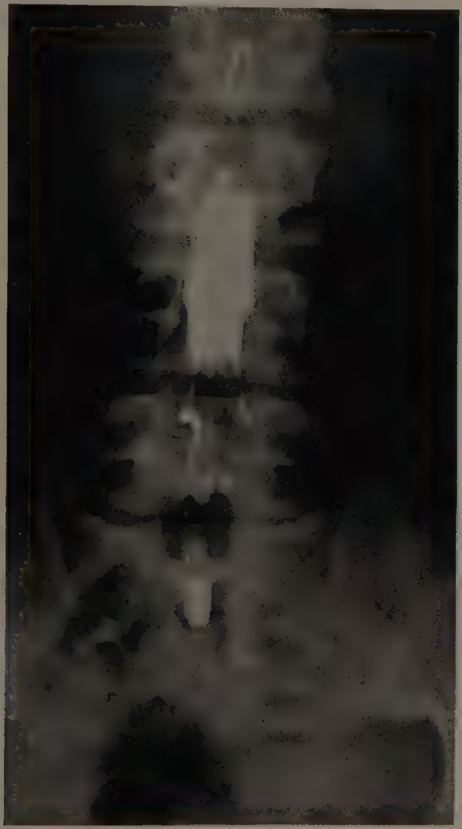

FIG. 29. R.R. No. 33813. Roentgenogram (patient erect) shows narrowing of the left side of the intervertebral disc between L 4 and L 5 due to posterior herniation of the nucleus pulposus. Lipiodol obstructed opposite this disc (arrows).

Intraspinal calcification adjacent to the dorsal margin of the disc. Calcification adjacent to the dorsal margin of an intervertebral disc is seen occasionally, and its presence signifies a posterior herniation of the nucleus pulposus which has undergone calcification. This mass of calcification is usually only a few millimeters in size and may be directly in the midsagittal plane of the spine, or off to one or the other side of the spinal canal. In the dorsal region such calcification in the ventral portion of the spinal canal may be associated with calcification of an intervertebral disc, and it is always

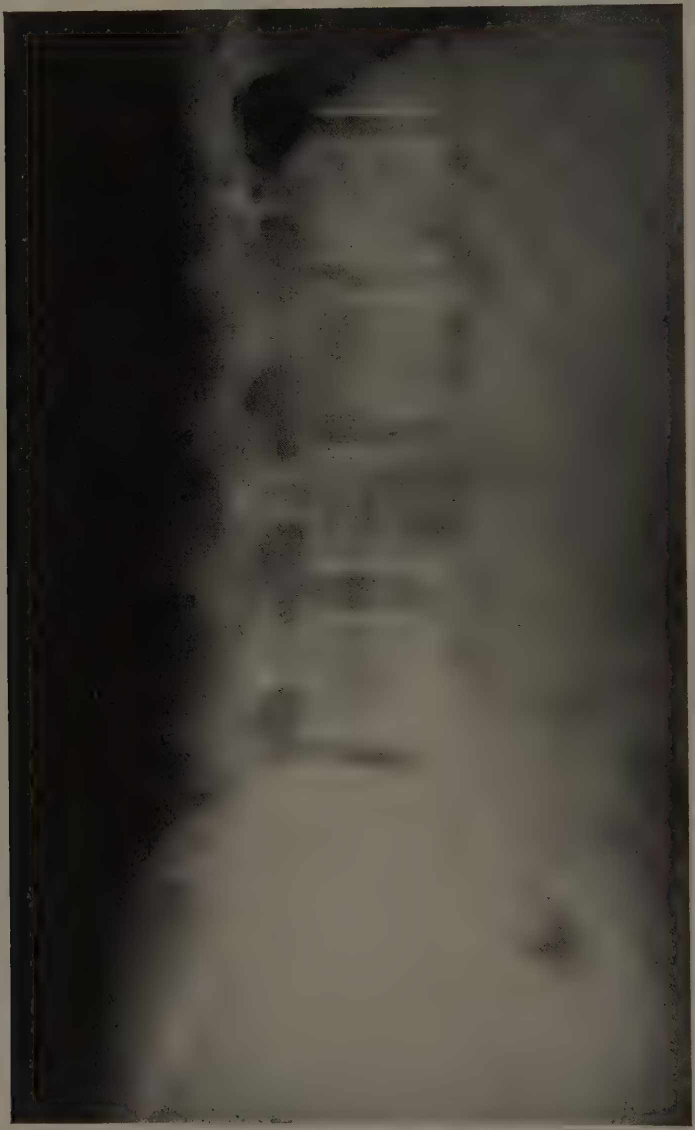

FIG. 30. R.R. No. 34886. Lateral view of the spine showing the obliteration of the normal lumbar lordosis associated with posterior herniation of the nucleus pulposus.

well to scrutinize the ventral portion of the spinal canal when such calcification of the intervertebral cartilage is noted.

Obliteration of lumbar lordosis. Obliteration of the normal lumbar lordosis is almost uniformly noted in patients who have a posterior herniation of the nucleus pulposus (Fig. 30). The degree of amelioration of the normal lumbar lordosis varies. In some instances it is only slight, while in others the lumbar spine is perfectly straight, and in rare cases, the lumbar spine has a slight ventral concavity.

Scoliosis. Scoliosis of the lumbar portion of the spine is commonly due to a posterior herniation of the nucleus pulposus in the caudal portion of the canal. The degree of scoliosis naturally varies from case to case. In some this may be eliminated temporarily by conscious effort, but in others the pain associated with the lesion is so great that the scoliosis cannot be eliminated. The margin of the intervertebral discs on the concave side of the scoliosis may be narrowed.

Localized spur formation at the posterior and lateral margins of the bodies of the vertebrae. Spur formation at the posterior margins of the bodies of the vertebrae adjacent to a posterior herniation of the nucleus pulposus occasionally is seen. The spurs seen in association with this lesion are directed mainly backward and are somewhat more removed from the margin of the disc than is usually the case in ordinary hypertrophic arthritis.

Lipiodolography

Barr and Mixter and their co-workers (1934) described the following technique for the identification of the posterior herniation of the nucleus pulposus. The patient is placed on his side and a spinal puncture performed, at which time 5 cc. or more of cerebrospinal fluid is withdrawn and 5 cc. of undiluted heavy lipiodol is introduced into the spinal subarachnoid space in the lumbar region. The patient is then placed in the prone position so that the lipiodol will come into contact with the dorsal margins of the vertebral bodies and the intervertebral discs (Fig. 31). With the patient in this position, much smaller masses projecting into the ventral spinal subarachnoid space may be identified than when the patient is in the supine position. In fact, sizeable posterior herniations of the nuclei pulposi may be entirely unrecognizable with the patient in the supine position (Fig. 32) and readily identifiable when the patient is in the prone position. Therefore, with the patient prone, the lipiodol is allowed to move slowly cephalically to the dorsal or mid-dorsal portion of the spinal subarachnoid space or even into the cervical

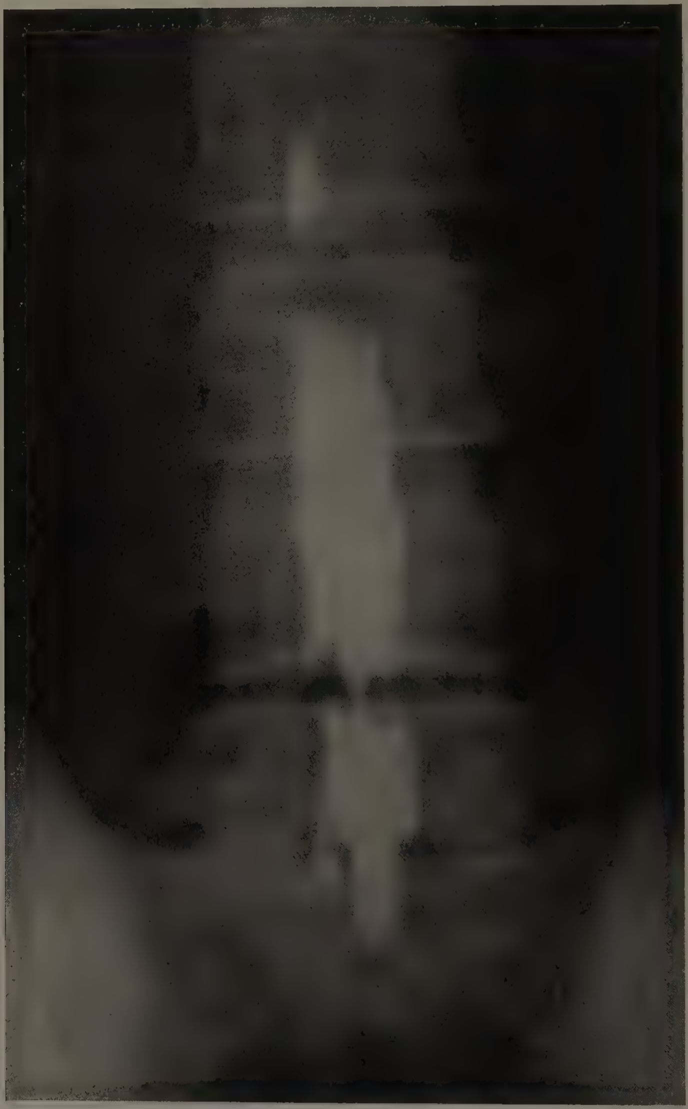

FIG. 31. R.R. No. 30965. Anteroposterior roentgenogram of the lower lumbar spine with the patient prone, showing the hourglass constriction of the lipiodol between L 4 and L 5 due to posterior herniation of the nucleus pulposus.

region, if it is indicated. Following this, the lipiodol is allowed to pass caudally through the lower portion of the lumbar subarachnoid space until it has reached the caudal end of this space, which generally

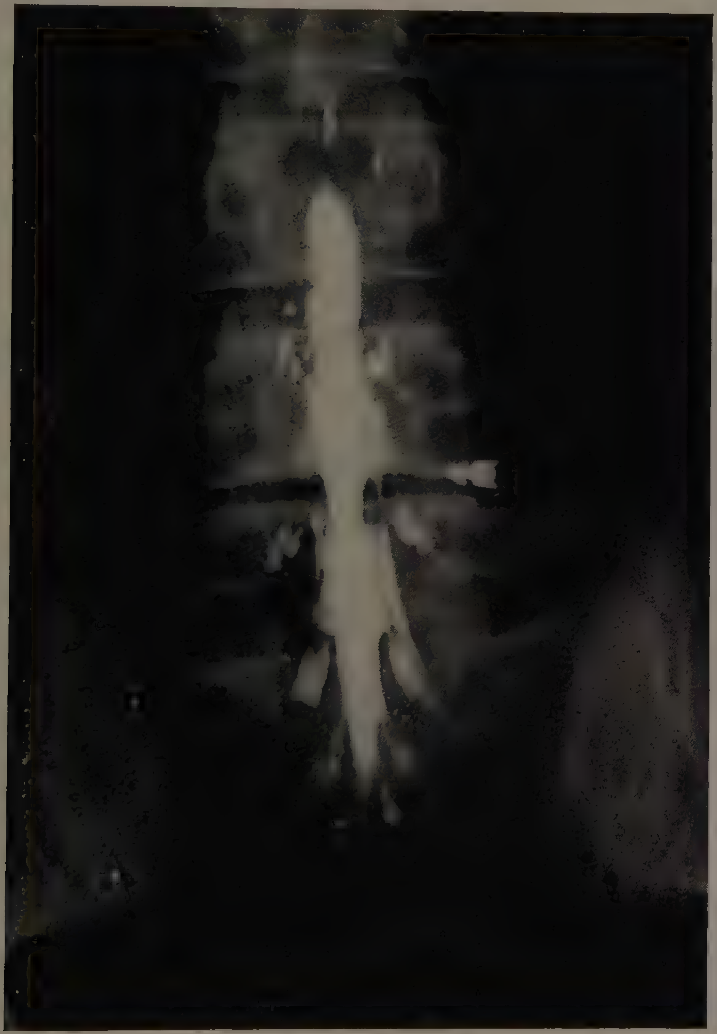

FIG. 32. R.R. No. 30965 (same case as Fig. 31). Roentgenogram made with the patient supine. Note that the filling defect in the lipiodol has practically disappeared.

is opposite the upper margin of the second sacral segment. This varies somewhat and in some cases the caudal end of the subarachnoid sac may extend no lower than the first sacral segment, but rarely is it more cephalic than this. As the lipiodol flows through the lumbar region it will be noted that opposite each intervertebral disc there is normally a slight symmetrical narrowing of the lipiodol.

If a herniation of the nucleus pulposus exists, the lipiodol usually deviates to the side opposite the mass and becomes pointed, and as it extends beyond the mass the lipiodol column again spreads out, so that a notch is formed by the intraspinal mass into the margin of the opaque medium. In many cases, the lipiodol, instead of proceeding to one side of the subarachnoid space around the mass, will be constricted almost evenly on the two sides so that the opaque medium will have a definite "hourglass" appearance. Furthermore, when the herniation occupies the mesial portion of the spinal subarachnoid space and is quite broad, the lipiodol may flow up to this bar and become obstructed, and only after elevating the cephalic end of the patient to a considerable degree will the lipiodol tumble over the obstruction. It is of the utmost importance that the patient be tilted up and down slowly so that the column of lipiodol does not break spontaneously.

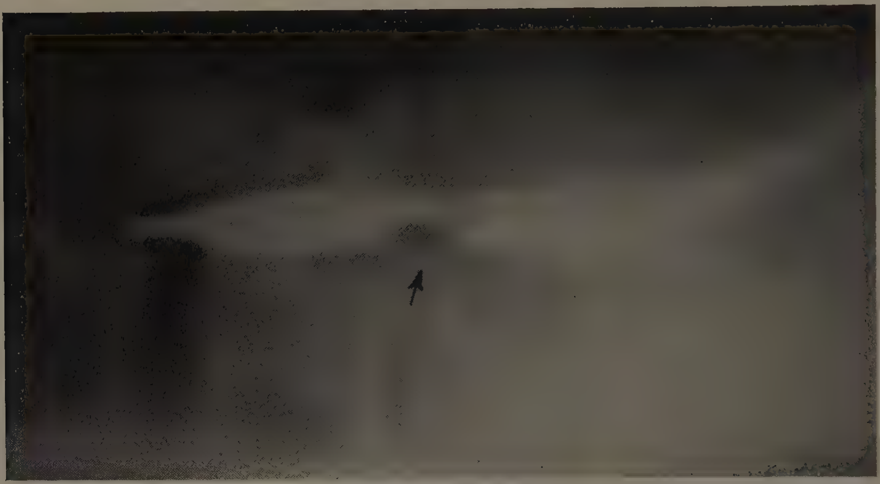

FIG. 33. R.R. No. 30965 (same case as Figs. 31 and 32). Lateral projection of the lumbar spine with the patient prone, showing the notch in the lipiodol opposite the disc between L 4 and L 5.

When the patient is fluoroscoped in the prone position, but with the tube at one side and the fluoroscopic screen at the other side of him, a slight notch due to the intervertebral disc is normally seen.

This amounts to about 1 mm. in the average case. When a herniation of the nucleus pulposus into the canal exists, the defect in the column of lipiodol as seen from the side or profile often amounts to from 5 mm. to 1 cm. (Fig. 33).

In about 10 per cent of the patients who have a posterior herniation of the nucleus pulposus, there is sufficient narrowing of the spinal subarachnoid space, even in the lumbar region where no spinal cord exists, to give a complete or practically complete block to the opaque medium. Posterior herniation of the nucleus pulposus into the spinal canal in the cervical region is considerably more difficult to visualize roentgenologically than one in the lumbar area. Frequently, however, in the cervical region, one notices some narrowing of an intervertebral disc and spur formation extending into the spinal canal. When the lipiodol investigation is made, the lipiodol extends around the mass as already described in the case of lumbar herniations.

Gas Myelography

Gas myelography was first attempted by Wideröe (1921) in a case of spinal cord tumor. Incidentally, when in his case the gas was not obstructed, he visualized it in the cranial cavities and believed himself the first person to perform encephalography. Since this initial experience of Wideröe numerous workers have employed gas to visualize masses projecting into the spinal subarachnoid space. Air may be used, although at the Neurological Institute it has been the routine practice to use oxygen, in the belief that it is somewhat less irritating to the spinal nerve roots. When a gas is employed instead of a radio-opaque medium, it is necessary to have the patient in such a position that the hips are higher than the shoulders. Usually 30 to 50 cc. of spinal fluid are withdrawn from the lumbar subarachnoid space and an equal quantity of gas is introduced in small amounts by alternating the removal of fluid and the injection of the gas. Visualization of the gas in the spinal canal is much more difficult than that of lipiodol, and therefore more errors are made in diagnosis than when lipiodol is utilized. However, gas has the advantage of being completely absorbed without inflammatory reactions occurring in the intraspinal tissues, such as have been ascribed to lipiodol.

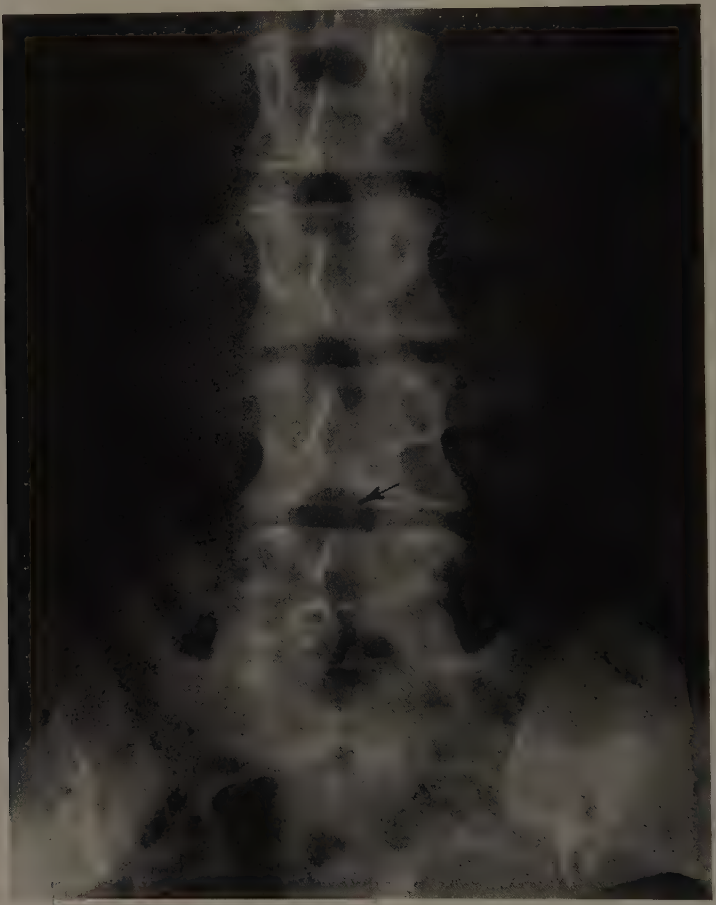

FIG. 34. R.R. No. 46921. Defect (arrow) in left side of column of gas opposite the disc between L 4 and L 5, due to posterior herniation of the nucleus pulposus.

When gas myelography is used, fluoroscopy is useless since one is unable to see the gas in this type of examination. The roentgenograms are made with the patient supine, which facilitates the accumulation and concentration of gas along the dorsal margins of the vertebral bodies and intervertebral discs. Roentgenograms are made stereoscopically with the patient's hips elevated at least 30 degrees from the horizontal (Fig. 34). In addition, the patient is usually turned on one or the other side and lateral roentgenograms are made. With the patient in the lateral position, the gas tends to approach the upper lateral margin of the canal and may fail to disclose a herniation of the nucleus pulposus.

Hypertrophy of the Ligamentum Flavum

The ligamentum flavum is an extradural ligament which extends between the pedicles and laminae on each side of the spinal canal. In certain cases, due to injury or disease, the ligament increases in thickness in a localized area. Such enlargement may be sufficiently great to encroach on the canal and impair the function of the nerve roots, thus producing a clinical picture quite indistinguishable from that of posterior herniation of the nucleus pulposus.

In the lipiodol examination the largest defect in the opaque medium will occur with the patient supine, which is the reverse of what occurs when the nucleus pulposus has herniated into the spinal canal. In certain instances, however, when the ligamentum flavum is thickened laterally opposite the pedicle of the vertebra, only a unilateral notch in the opaque medium occurs. In such cases, it is impossible to differentiate between a posterior herniation of the nucleus pulposus and thickening of the ligamentum flavum. In the lateral view, the dorsal margin of the lipiodol is encroached upon, and this is of aid in the differential diagnosis.

In a number of patients who show a definite encroachment on the spinal subarachnoid space, as indicated by notching or narrowing of the column of lipiodol particularly in the lower lumbar area, no herniation of the nucleus pulposus or thickening of the ligamentum flavum can be found at operation. There is no entirely satisfactory explanation for some of these cases, but in others the deformity of the lipiodol is probably due to hyperplasia of extradural fat or anomalous blood vessels. That the diagnosis of encroachment by a mass on the spinal subarachnoid space probably is correct, is suggested by the fact that a laminectomy will relieve the symptoms of many of these patients. In other words, the decompression produced by the laminectomy apparently is sufficient therapy to obtain a satisfactory result. Another explanation for the surgeon's inability to find an intraspinal mass corresponding to the defect in the lipiodol, as seen in the roentgenograms, may be due, as Chamberlain* and Fay have suggested, to the position of the patient on the operating table. It is customary to have the spine somewhat flexed to facilitate performing the laminectomy and this may cause the herniated nucleus to disappear within the disc.

GENERAL ROENTGEN RAY SIGNS OF SPINAL CORD TUMORS

Tumors of the spinal cord and its membranes frequently produce changes in the vertebrae and soft tissues which indicate their site.

In the past, roentgenographic examination has played a minor rôle in the localization of these tumors, but now it is rapidly becoming of major importance. In order to diagnose these tumors from the roentgenograms, it is essential that the examiner have a thorough knowledge of the size and shape of the spinal canal and the appearance of the individual parts of the vertebrae. As these vary in the different portions of the spinal column, the normal appearance of the various portions of a vertebra as they appear in the roentgenogram will be discussed. It is proposed in this discussion of spinal cord tumors to enumerate the points of practical value in making a diagnosis of these neoplasms. It is my purpose to consider mainly those neoplasms with which I have had personal experience over a period of years at the Neurological Institute of New York.

It is a fact not generally appreciated, that frequently the character of the growth and its location in relation to the cord, whether it is intramedullary, extramedullary or extradural, can be determined from an examination of the roentgenograms. It is well to point out that spinal cord tumors, like brain tumors, do not metastasize to distant organs of the body. Furthermore, spinal cord tumors produce changes in the vertebrae by pressure rather than by infiltration, with the exception of the rare tumor known as chordoma.

In order to properly evaluate roentgenograms in which spinal cord tumors appear, it is necessary to know certain clinical facts regarding the behavior and signs produced by each type of spinal cord tumor. These data regarding the incidence of certain tumors, their location in relation to the spinal canal and meninges, and their frequency of occurrence as to sex, age, and so forth, will be found in another section of this book.

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* Personal communication.

Plain Roentgenograms (Flat and Stereoscopic)

Tumors of the spinal cord and its coverings, and also of the nerve roots, do not produce a reaction on the vertebrae distant from the site of the lesion. In this way they behave differently from tumors found within the intracranial cavity. Spinal cord neoplasms often cause changes in the contiguous vertebrae, and may change the surrounding soft tissues over a localized area corresponding to the site of the growth. Because of this, it is frequently possible to localize the lesion at a specific vertebral level and to determine its size. In a number of instances, the histologic nature of the tumor is not evident after a careful study of the roentgenograms, but there are a number of cases in which the exact type of tumor may be diagnosed.

The factors which must be noted in the roentgenograms and upon which a diagnosis is based are: (1) Bone destruction, (2) appearance of and measurement between the pedicles, (3) distortion of the paraspinal soft tissues, (4) bone proliferation, (5) abnormalities of the intervertebral discs, (6) kyphosis and scoliosis, and (7) calcification. These factors have been enumerated in accordance with their importance.

Bone Destruction. Bone destruction is the most common change produced in the vertebrae by a tumor of the spinal cord or by one arising in the meninges. In the case of primary tumors arising in the spinal cord or in the meninges, the bone is destroyed due to pressure of the mass against the resistant and unyielding bone. This results in localized pressure atrophy of the bone contiguous to the tumor, and soon the bone becomes decalcified, appearing less dense than normal in the roentgenogram. However, continuous pressure leads to varying degrees of destruction in one or more portions of one, two, or, in rare cases, several vertebrae. In certain instances, when the pressure on the osseous structures has been prolonged, complete destruction of one or more pedicles of a single vertebra or of two vertebrae occurs. The pressure usually is marked in the pedicles, and destruction of this portion of the vertebra is the easiest to recognize in the roentgenogram. This is partly due to the fact that the pedicles are more satisfactorily visualized in the roentgenogram than are some other parts of the vertebra, and smaller changes can be detected. Furthermore, the pedicles have a fairly constant size, shape and density, which also aids in the recognition of smaller changes here than when other portions of the vertebra, such as the body and laminae, are affected. The spinous process is infrequently involved.

Another osseous structure which may be affected by a spinal cord tumor is the rib, or in certain cases, several ribs. The portions of the rib involved are: the head, neck, and the tubercle where the rib articulates with the transverse process of the vertebra. Occasionally a part of the shaft of one or more ribs may be partially destroyed, or there may even be a dislocation of the rib due to pressure of the tumor as it grows between them.

It is important to remember that true spinal cord tumors do not actually invade the surrounding bone but produce atrophy which is dependent upon continued pressure. The atrophy is accelerated by the pulsation of the mass against the osseous structure. The pulsation of the mass is due to transmitted pulsation from the other spinal contents.

Appearance of Pedicles. In the great majority of cases the correct diagnosis as to location, extent, and type of tumor depends upon the appearance of the pedicles of one or more vertebrae, and so it is advisable to describe their normal appearance in the various portions of the spine. The pedicles are more satisfactorily visualized in the anteroposterior roentgenograms, but they are also clearly outlined in the lumbar and cervical regions. In the dorsal region of the lateral roentgenograms, the pedicles are somewhat more difficult to visualize due to the complexity of the superimposed shadows, namely, ribs and intrathoracic contents. Furthermore, it is of importance to emphasize that oblique films of the spine often give information of the greatest value in the diagnosis, for in the oblique view the pedicles and also the intervertebral foramina may be clearly visualized. In fact, this is the only position in which the intervertebral foramina are satisfactorily outlined.

In the roentgenograms taken in the anteroposterior view, the shape of the pedicles is usually more or less oval with the long diameter vertically placed. However, there are many exceptions to this statement; for example, in the cervical region the pedicles appear narrow, while in the lumbar region they are almost circular. In the cervical region, the mesial border of each pedicle changes from flat to slightly convex, while in the dorsal and lumbar segments of the spinal column, the medial borders of the pedicles are usually convex, although there are frequent exceptions. The frequency with which these exceptions occur in any part of the spinal column makes it unwise to base a diagnosis of a spinal cord tumor solely upon the existence of flat or concave pedicles. It is true, however, that pedicles with concave medial borders are more frequent at the site of a spinal cord tumor than in a normal spinal column, but the exceptions are too frequently observed to consider flat or concave pedicles pathognomonic of a spinal cord tumor. In addition to the change in the curve, there must be thinning of the cortical bone forming the mesial border of the pedicle, if a diagnosis of tumor is to be made. It is well to point out that in children the medial borders of the pedicles, cephalad to the seventh dorsal vertebra, are commonly concave medially, but in adults this is rare unless a pathologic process is present. Thus it is advisable to state that, in adults, a concave mesial border of a pedicle cephalad to T 7 is suggestive evidence of a pathologic lesion at the segment where the concave pedicle occurs.

Measurements between Pedicles. The interpediculate distance is the space between the most mesial points of the pedicles of a single vertebra. Roentgenograms of the spine taken in the anteroposterior position are most useful for determining this distance, that is, the width of the spinal canal. Elsberg and Dyke disclosed that the width of the vertebral canal can be accurately determined from the roentgenogram by measuring the shortest distance between the medial borders of the pedicles of each vertebra. The interpediculate distance is fairly constant for each segment, but since there are variations, it is well to use moderate caution in the interpretation of enlargement of this distance. Generally speaking, the more localized the enlargement of the spinal canal, that is, the interpediculate distance, the more likely it is that it is due to a pathologic process. Occasionally, if a number of vertebral segments in the upper dorsal region are larger than the maximum normal the condition may be due to a developmental anomaly or anomalies and not to tumor. Furthermore, the interpediculate distance in the cervical region may be increased if the patient has a marked anterior convexity of the cervical spine, so that the distance from the vertebrae to the film is considerably greater than usual. A chart has been prepared by Elsberg and Dyke which gives the average and the extreme measurement for the interpediculate distance of any vertebra from the

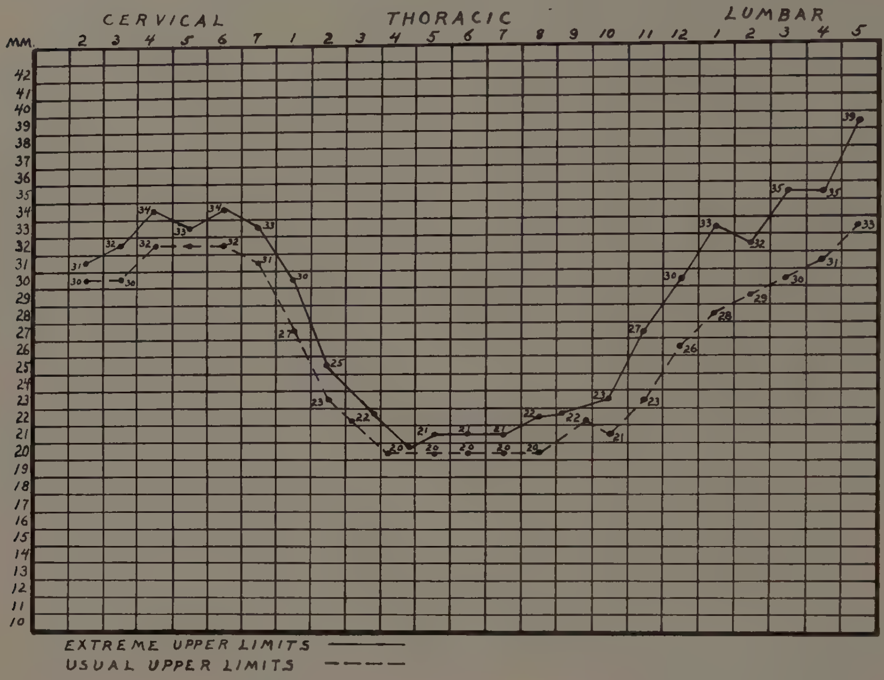


FIG. 35. Chart showing extreme (solid line) and average (broken line) interpediculate measurements of each vertebra in millimeters, from C 2 to L 5, inclusive.

second cervical to the fifth lumbar inclusive, in patients over 12 years of age (Fig. 35). The chart is based on an examination of 300 normal cases, and makes it possible to compare the measurements of any given case with a normal standard; up to the present time it has proved of considerable value. However, in medicine there are exceptions to every rule, some of which I have already mentioned. It should be emphasized that the interpediculate distance is only one of the criteria to determine whether or not a spinal cord tumor exists, and if an abnormal measurement is associated with slight atrophy of one pedicle or with a small paraspinal soft tissue mass, it is of greater value than if only a slight increase in the interpediculate distance alone exists.

Upon examination of the chart it will be seen that the vertebral canal is wide in the cervical region. From either the seventh cervical or the first thoracic segment, the width of the spinal canal decreases rapidly to the fifth thoracic vertebra. From this point caudally, the width of the vertebral canal remains quite constant to and including the ninth dorsal vertebra. From the tenth dorsal vertebra to the fifth lumbar, the width of the canal usually increases in size.

Tumors of the spinal cord and its coverings often produce a localized enlargement of the spinal canal. When, therefore, the interpediculate measurements are greater than the maximum normal, they are of real significance. Smaller measurements within the normal have no importance, but when a relative enlargement of the canal does occur, it, too, is of real significance. By the latter is meant a localized increase of 2 to 4 mm. or more in the distance between the pedicles of one or several vertebrae, where normally the width of the canal should remain the same or become progressively smaller or larger, as it does in the cervical and lumbar regions, respectively. However, the interpediculate measurements are within the extremes (Fig. 36).

Furthermore, the size of the tumor can often be foretold when the canal is accurately measured. Thus, if the interpediculate distance of only one vertebra is increased above the normal, the tumor is probably a small one, but if the interpediculate measurements of a number of vertebrae are increased, the tumor necessarily is large. Some types of tumors produce an enlargement of the canal of only one or two vertebrae, while other types affect three or more vertebrae. This fact makes it possible, in many instances, to determine the probable pathologic nature of the growth from the roentgenographic examination.

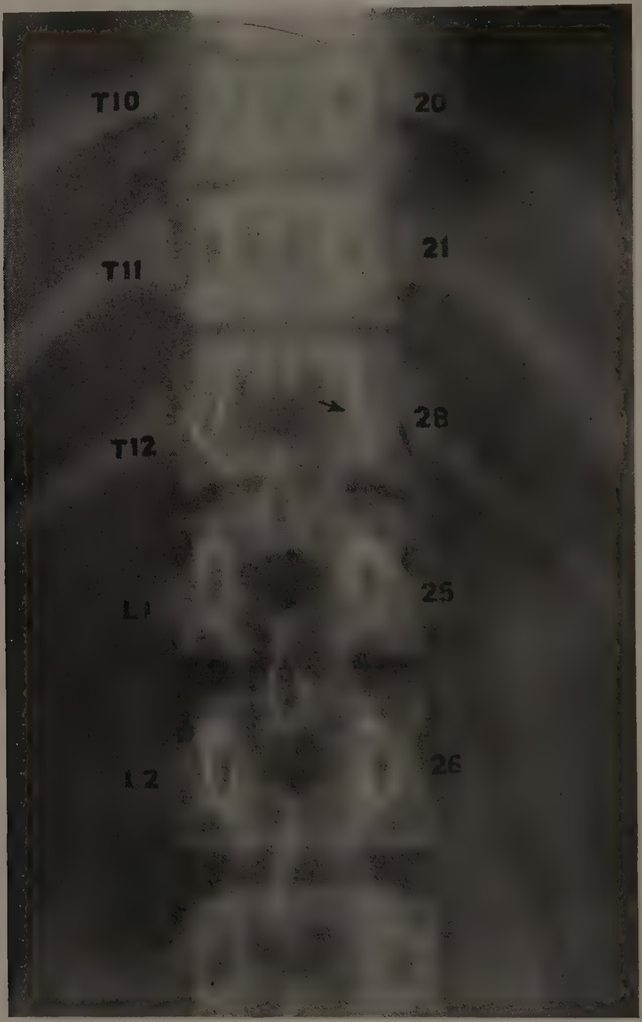


FIG. 36. R.R. No. 18540. Anteroposterior view of the lower thoracic and upper lumbar portions of the spine, showing slight flattening of the pedicles (arrow) and a relative increase in the interpediculate measurements of T 12.

Distortion of Paraspinal Tissues. The paraspinal tissues are represented by a linear soft-tissue shadow which parallels the spinal column on either side and is best seen on the left. These soft tissues are best outlined in the thoracic region on the anteroposterior roentgenogram. Normally the width of the vertebral paraspinal tissues varies in patients and does not appear to have any relation to their age, size, or sex. However, the shadow of these tissues always has a fairly straight lateral margin. This is important because primary intraspinal tumors frequently project through the spaces between adjacent vertebrae, and displace the tissues laterally. When this happens, a localized bulge in their contour is visible in the roentgenograms.

Bone Proliferation. Bone proliferation is rarely produced by spinal cord tumors. However, there is one form of growth (hemangioma) affecting the bone and meninges which causes the vertebrae to be denser than normal. The roentgen evidence of this lesion will be discussed under that heading. The tumors which arise in the bone, such as osteoma, osteochondroma, and osteogenic sarcoma which is rare, are naturally excluded from the above statement.

Osteo-arthritis, either localized or generalized, has no value in the diagnosis of spinal cord tumor. When it occurs in the presence of an intraspinal neoplasm, it undoubtedly is a coincidence and not the result of the tumor.

Abnormalities of the Intervertebral Discs. Abnormalities of the intervertebral discs are rarely produced by spinal cord tumors. The resilience of the cartilage is an important factor in its preservation, even though sufficient pressure is being exerted to produce considerable destruction of portions of the adjacent vertebrae. Furthermore, destruction of the intervertebral cartilage in the roentgenogram is difficult to visualize, since the cartilage itself cannot be seen, and its loss can be determined only if the intervertebral space is narrowed.

Calcification within an intervertebral disc may have clinical significance, as shown in several cases of ventral chondroma where areas of calcification within the disc were visible at the level of the tumor. Other spinal cord tumors do not produce this type of reaction in the intervertebral cartilage.

In two cases of ecchondrosis, narrowing of a single intervertebral disc was noted at the level of the lesion.

Kyphosis and Scoliosis. These two conditions may be considered together. Kyphosis of the spine at the site of a spinal cord tumor is sometimes seen. This has been noted in the cervical region in association with several tumors. Unless there is localized bone destruction, its significance remains doubtful.

Coexistence of a lateral curvature of the spine and a neoplasm of the spinal cord is occasionally seen. However, scoliosis in conjunction with a tumor is probably a coincidence rather than a result of the new growth. Furthermore, no part of the curve seems to bear a constant relation to the site of the neoplasm and consequently is of no localizing value.

Calcification in Tumor. A spinal cord tumor containing calcification within the spinal canal in sufficient amounts to be seen in the roentgenogram is infrequent. However, such calcification may occur; and when a definite irregular mass of lime is seen within the spinal canal, the location and type of the tumor have been determined, for in this clinic such an irregular mass of calcification has always proved to be within a meningioma. It is probably also worthy of mention that not infrequently meningiomas contain microscopic deposits of lime, and this fact combined with the continued improvement in roentgen technique may lead to the roentgen diagnosis of a greater number of these tumors in the future.

ROENTGEN RAY EVIDENCE OF SPECIFIC TYPES OF SPINAL CORD TUMORS

Intramedullary (Chapters vii, viii, ix)

Lipoma. Lipomas arising within the substance of the spinal cord are extremely rare. However, they may produce atrophy of the surrounding bone. These tumors are usually encountered in patients in the first or second decade of life. The neoplasm is most often found in the lower cervical or upper dorsal regions of the spinal cord and extends over a number of vertebral segments. As in most spinal cord tumors, the fundamental change noted in the roentgenogram is erosion of portions of the vertebrae, and usually three or more vertebrae are involved. This fact is important because there are only a few tumors which affect more than two vertebral seg-

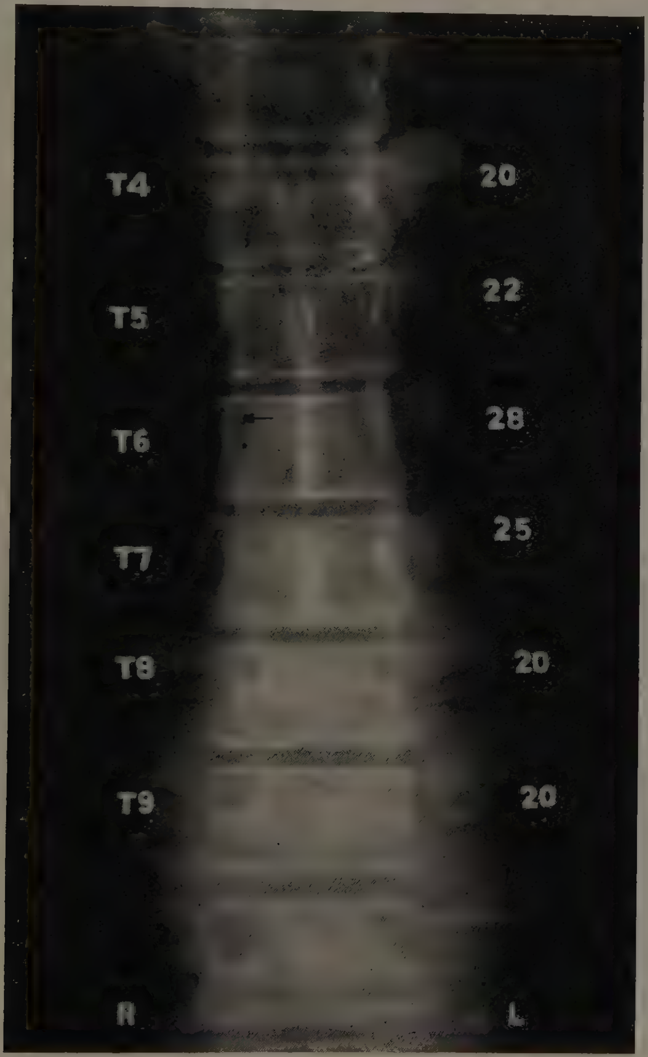


FIG. 37. R.R. No. 28068. Intramedullary lipoma. Anteroposterior view of the upper thoracic spine, showing flattening of the pedicles of a single vertebra (arrow) and an increase in the interpediculate measurements of the fifth, sixth, and seventh thoracic vertebrae. The actual distance between the pedicles in millimeters is given to the left of each vertebra.

ments. In addition to the lipomas, the congenital cysts, ependymomas, and the large unclassified tumors of the cauda equina are also likely to produce destructive changes in a number of vertebrae. Figure 37 is a reproduction of a roentgenogram of the dorsal portion of the spine showing the changes produced by a lipoma.

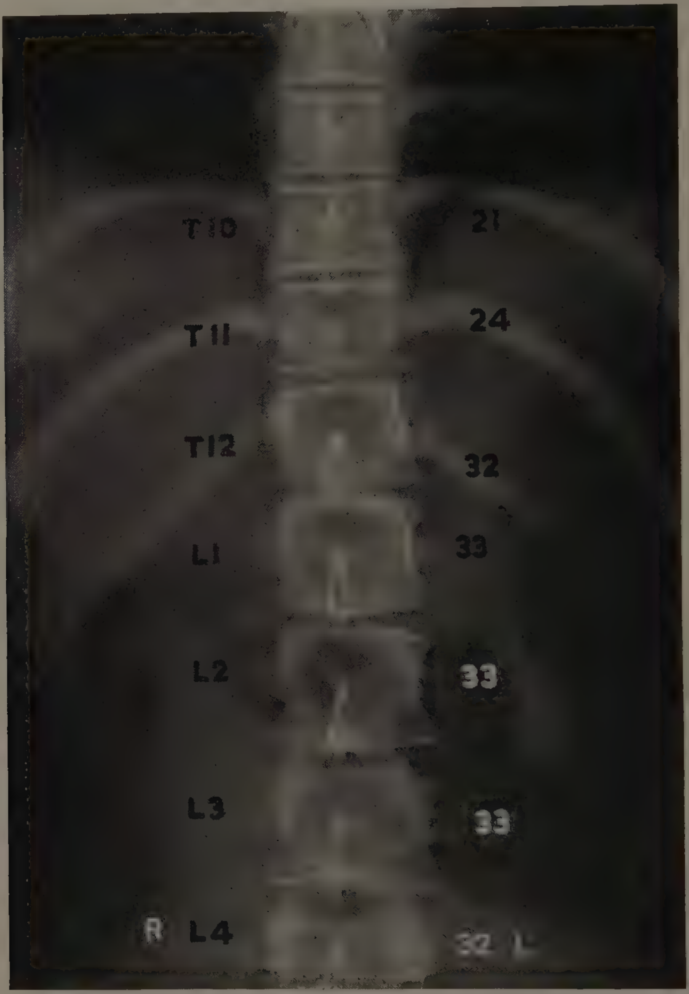


FIG. 38. R.R. No. 29028. Ependymoma of filum terminale. The film shows especially well the increase in the interpediculate measurements, and the flattening of the pedicles from T 12 to L 3, inclusive.

Ependymoma. The ependymomas are common glial tumors which occur in adults, particularly young adults, and usually originate in the filum terminale or conus medullaris. This type of tumor is extremely slow growing and many of the patients harboring such a growth do not come under observation until the growth has attained a large size. The slow growth and large size cause bone changes in a high percentage of cases. In many instances extensive destruction of a number of vertebrae is present (Figs. 38 and 39). The destruction of the bone is due to pressure erosion and not to infiltration by tumor tissue. Characteristically, four or more vertebrae are involved and

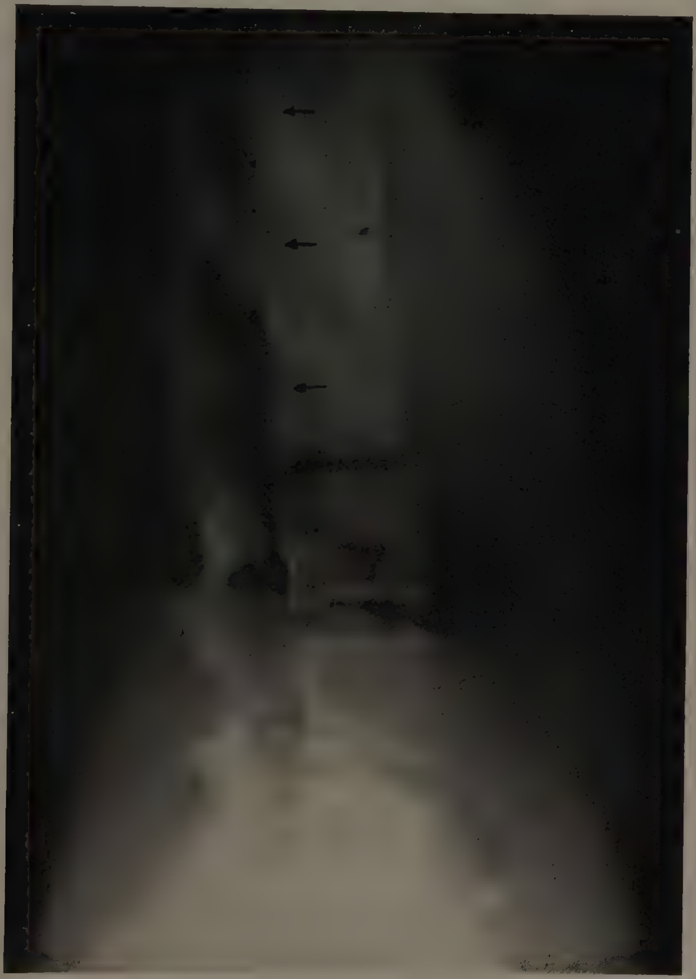


Fig. 39. R.R. No. 29028 (same case as Fig. 38). Lateral view of lower thoracic and lumbar portions of the spine. This shows the hollowing of the posterior surfaces of the vertebral bodies and the thinning of the pedicles. The anteroposterior diameter of the canal is increased.

the bodies are affected in the vast majority of cases. The posterior margins of the bodies of several vertebrae are concave due to the pressure of the tumor. The greatest concavity is at the mid point between the cephalic and caudad margins of each body. This is so, because the intervertebral discs, due to their elasticity, resist the pressure of the tumor; and this protection extends to the adjacent portions of the vertebral bodies, but progressively decreases away from the intervertebral discs (Figs. 40, 41, 42, 43, 44, 45).

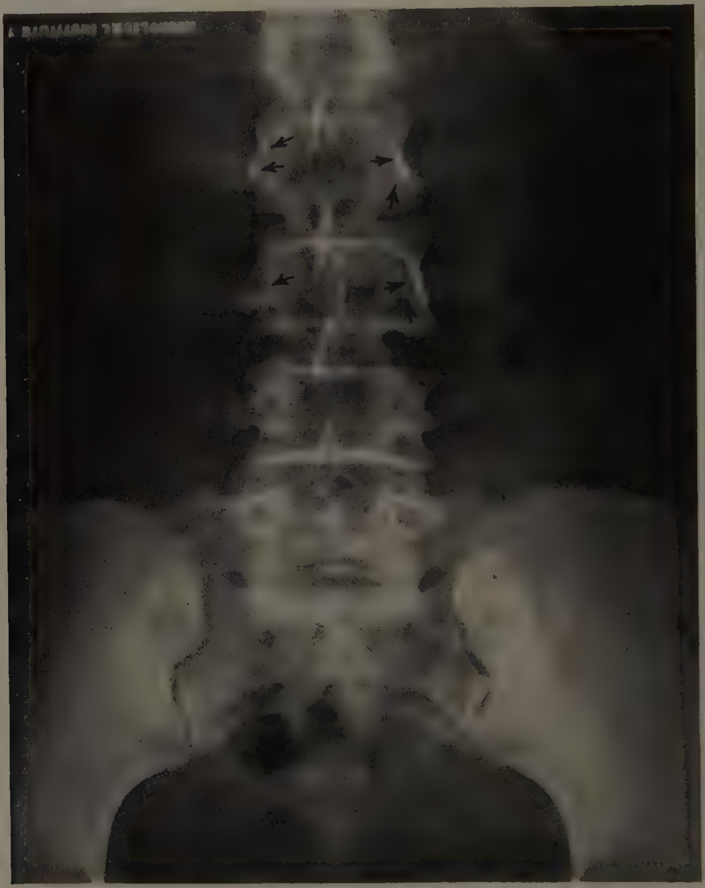


FIG. 40. R.R. No. 46213. Ependymoma. Anteroposterior roentgenogram showing marked destruction of the pedicles (arrows) and erosion of the dorsal portions of the bodies of L 2 and L 3.

The pedicles of a number of the vertebrae, as seen in the anteroposterior roentgenograms, are flattened and they may be concave.

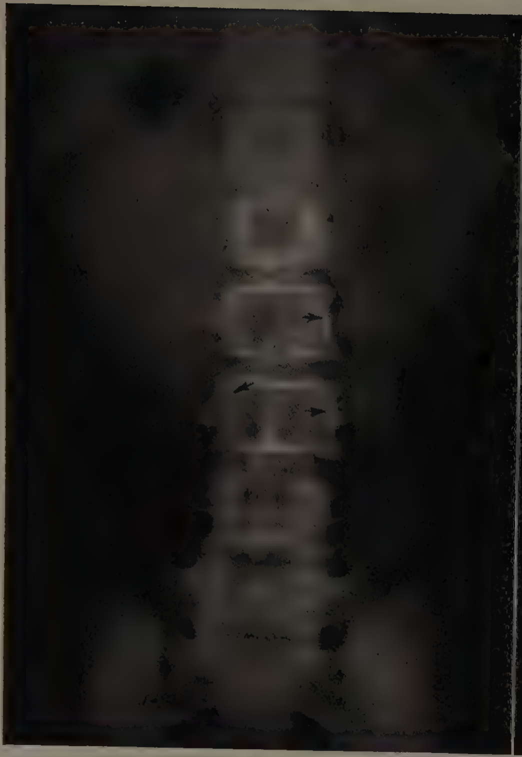


FIG. 41. R.R. No. 46312 (same case as Fig. 39). Laminagram showing the flattening of the pedicles and the pressure erosion of the dorsal portions of the bodies of L 2 and L 3. The section was made at a distance of 9 cm. from the table, with the patient supine.

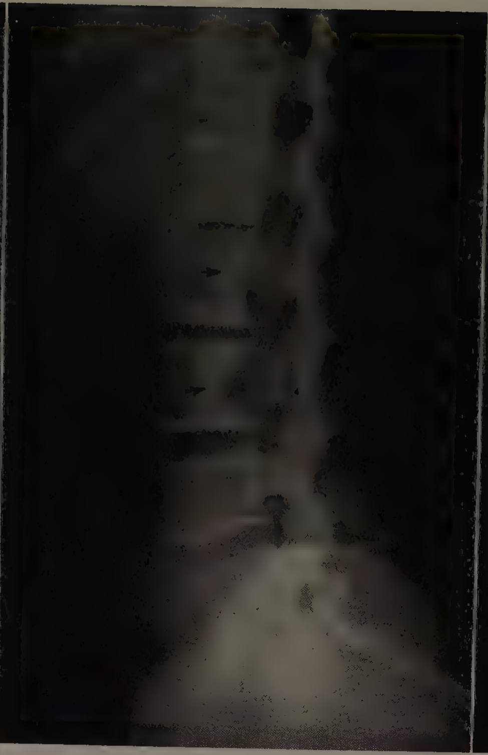


FIG. 42. R.R. No. 46213 (same case as Figs. 40 and 41). Lateral view showing marked erosion of the dorsal portions of the bodies (arrows) of L 2 and L 3, and marked thinning of the pedicles.

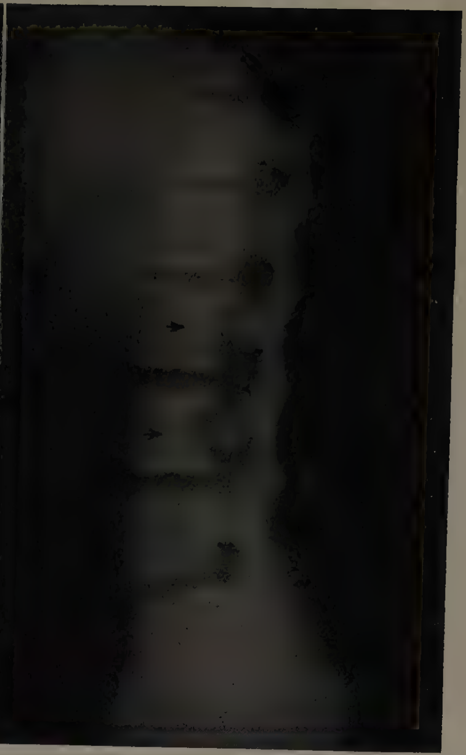


FIG. 43. R.R. No. 46213 (same case as Figs. 40, 41, and 42). Sagittal laminagram made at a distance of 11 cm. from table, showing erosion of the bodies (arrows) and pedicles of L 2 and L 3.

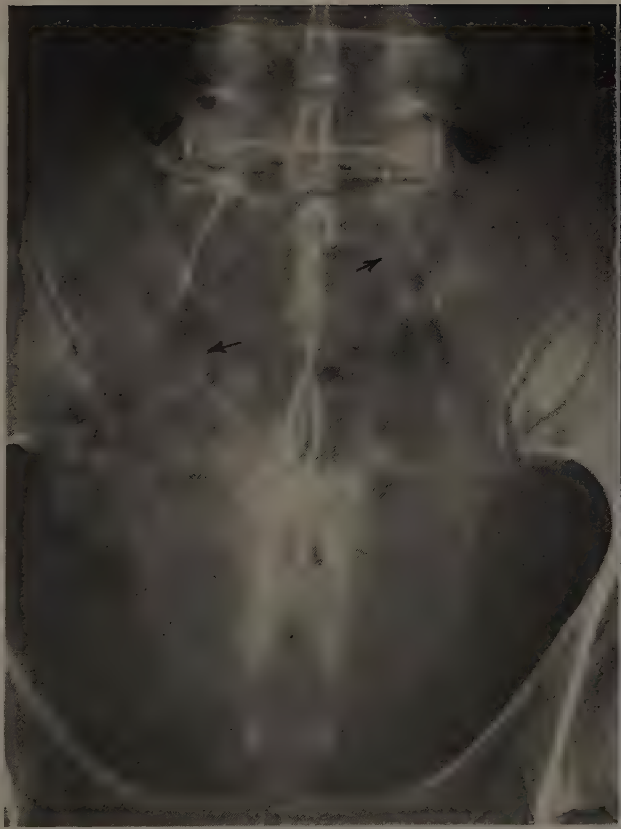


FIG. 44. R.R. No. 37537. Ependymoma causing expansion and pressure atrophy primarily of the first and second sacral segments (arrows). It also produced some concavity in the dorsal margins of the bodies of L 4 and L 5.

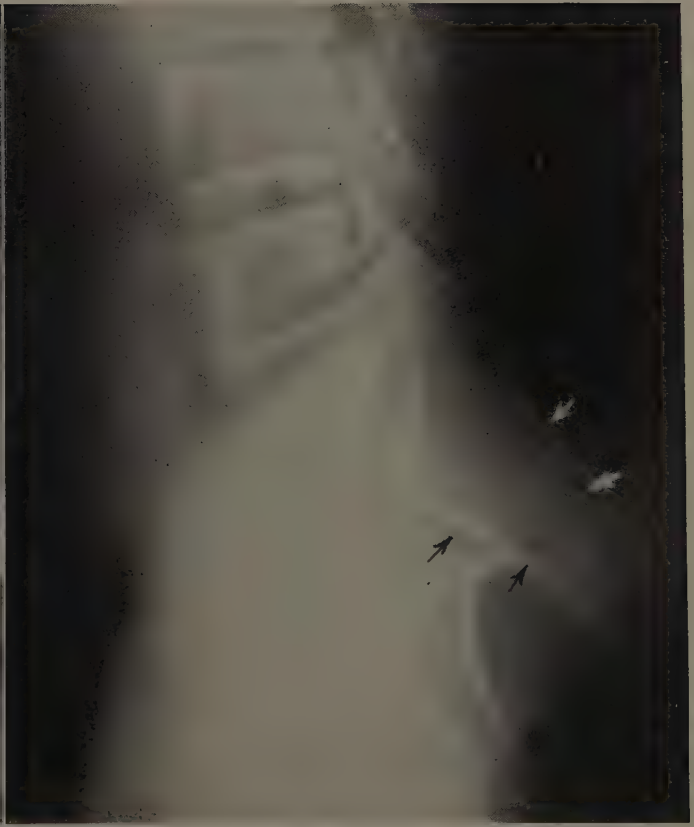


FIG. 45. R.R. No. 37537 (same case as Fig. 44). Lateral view showing expansion of the first and second sacral segments, and the concavity in the dorsal margins of L 4 and L 5.

Measurements of the interpediculate distances usually reveal an absolute enlargement over a number of segments. In the lateral view, the pedicles may be obscured due to the marked erosion. It is to be emphasized that when enlargement of the spinal canal with erosion of many pedicles and of the posterior margins of several vertebrae occurs in the lumbar region, a diagnosis of ependymoma is justified.

To summarize: The intramedullary tumors, such as spongioblastoma unipolare and multiforme, astroblastoma, oligodendroglioma and medulloblastoma infrequently produce changes in the adjacent parts of the spine, and therefore, with the exception of the ependymomas, are only occasionally detected in the roentgenogram.

Extramedullary Intradural Tumors (Chapter VII)

These neoplasms form 60 per cent of all spinal cord tumors and frequently produce changes in the vertebrae. This makes it possible to localize them in about 25 per cent of the cases. Furthermore, certain of these tumors are more prone to cause changes in the vertebrae than are others, as will be seen in the following paragraphs.

Meningeal Fibroblastoma (*Meningioma*). It is well known that certain tumors in various parts of the body have sites of predilection. This also is true for some of the spinal cord growths and especially for the extramedullary meningeal fibroblastomas (page 227). In about 8 per cent of the cases there is erosion of a portion of a single vertebra that is visible in the roentgenogram, which makes a localizing diagnosis possible. The destruction of bone is usually slight and only a single pedicle may be involved. It merely consists of thinning of the cortex of the bone forming the medial margin of a pedicle, but more commonly there is also, in addition to the latter, a flattening or concavity of its medial margin (Fig. 46). Moreover, if the distance between the medial borders of the pedicles is measured accurately, a relative increase will occasionally be noted (Fig. 36). The extramedullary meningiomas often contain microscopic deposits of calcium, and occasionally contain a sufficient amount to be visible in the roentgenogram. The calcium may consist of numerous flecks forming a cloudlike mass (Figs. 47, 48). This appearance, so characteristic of the meningioma, is due to countless small deposits of lime in the whorls of cells (Figs. 49, 50). In other cases, the calcium may form an irregular mass which is sharply demarcated and has the appearance of a small piece of bone. Thus, whenever macroscopic deposits of calcium occur within the spinal canal, it is likely that the lesion is a meningioma.

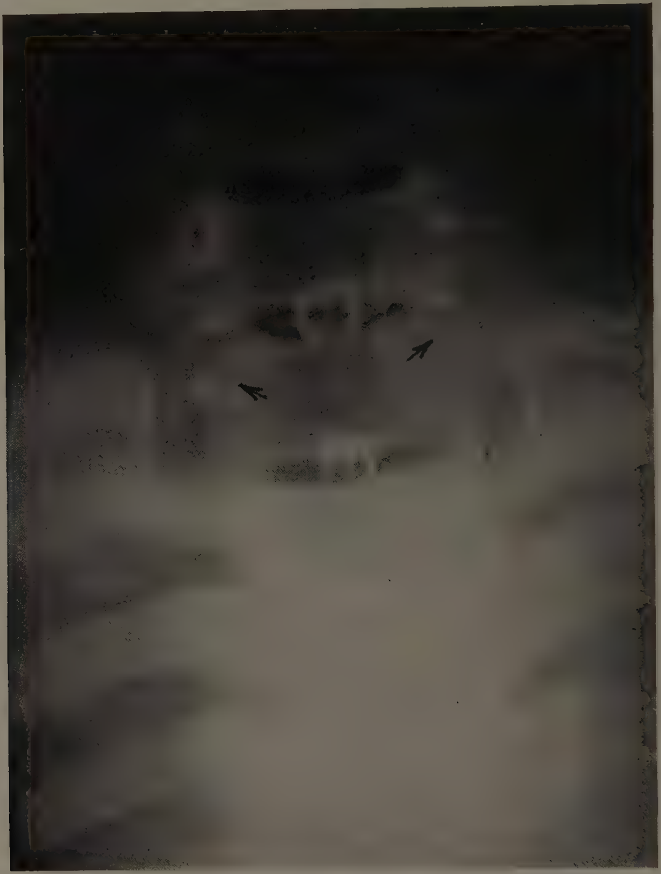


FIG. 46. R.R. No. 46488. Extramedullary intradural meningioma at T 1. The roentgenogram shows slight pressure erosion (arrows) of the medial margins of the pedicles of T 1.

Perineurial Fibroblastoma (*Neurofibroma*). The perineurial fibroblastoma is the second most common extramedullary tumor involving the spinal cord, and the one which most often gives roentgen ray evidence of its presence. It is found with equal frequency in the

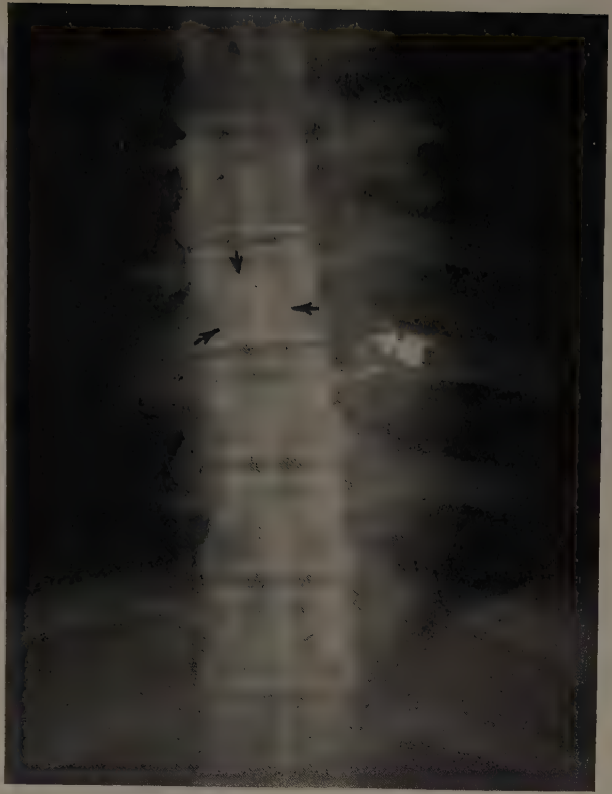


FIG. 47. R.R. No. 30327. Meningioma at T 6. Roentgenogram shows a globular shadow of increased density superimposed on the shadow of the sixth thoracic vertebra (arrows). Lead arrow indicates site of intradermal dye.

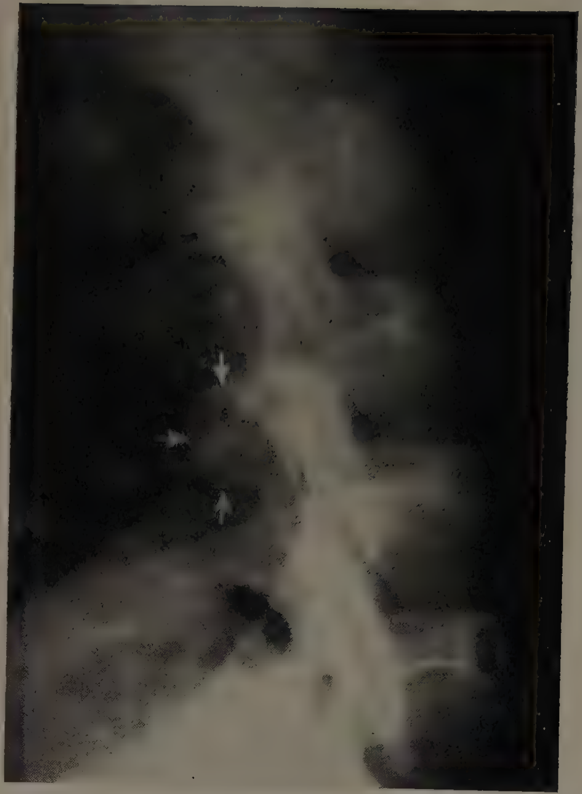


FIG. 48. R.R. No. 30327 (same case as Fig. 47). Lateral roentgenogram showing the globular tumor (arrows). The tumor is visible due to the many small areas of calcification in the whorls.

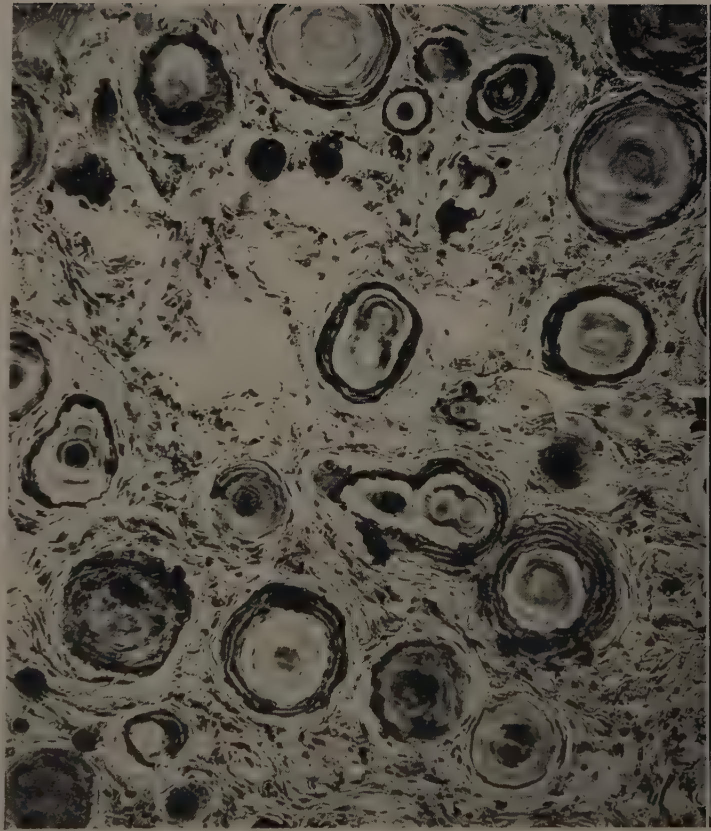


FIG. 49. Reproduction of histologic character of the meningioma portrayed in the roentgenograms in Figures 47 and 48. Note the large number of whorls with the flecks of calcium within them.

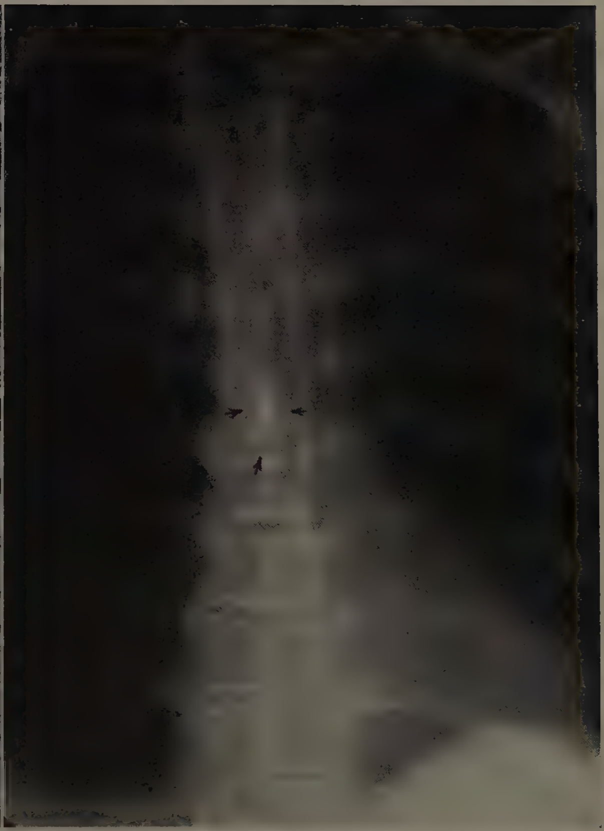


FIG. 50. R.R. No. 48506. Meningioma at T 6, showing the irregular calcification (arrows) in the tumor.

cervical, thoracic, and lumbar regions, if the relative number of vertebrae in each segment is taken into consideration. The predominating change produced in the vertebrae is bone destruction. This varies from slight erosion of a pedicle or lamina to extensive absorption. Slight or moderate atrophy of the medial margins of the pedicles of a single vertebra and flattening or concavity in the mesial border of the affected pedicles are the usual findings (Fig. 51). Not infrequently the pedicles of two vertebrae are affected. In the pres-

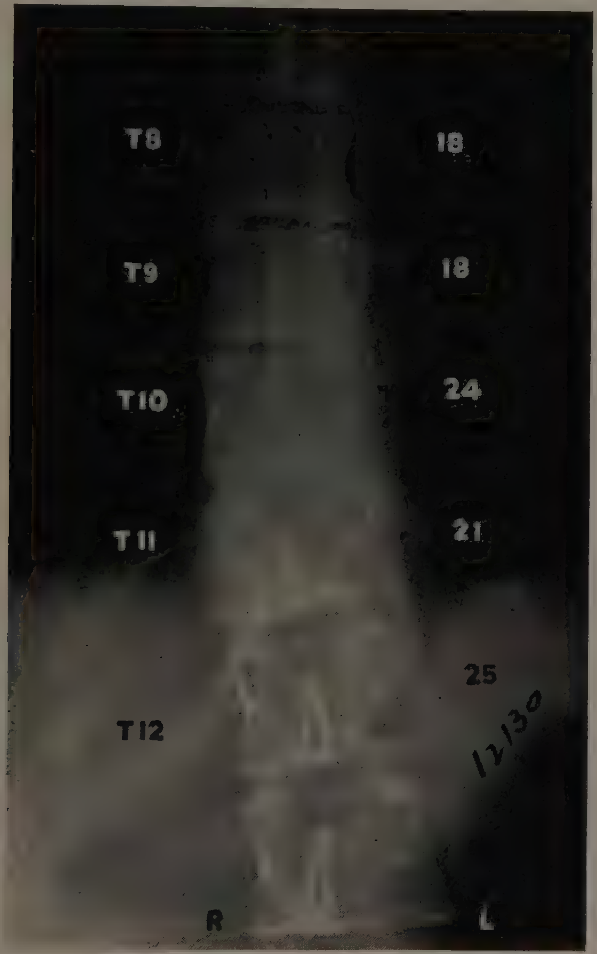


FIG. 51. R.R. No. 12130. Extramedullary neurofibroma at T 10, producing pressure atrophy of the medial and inferior margins of the pedicles at this segment. The interpediculate measurements are increased.

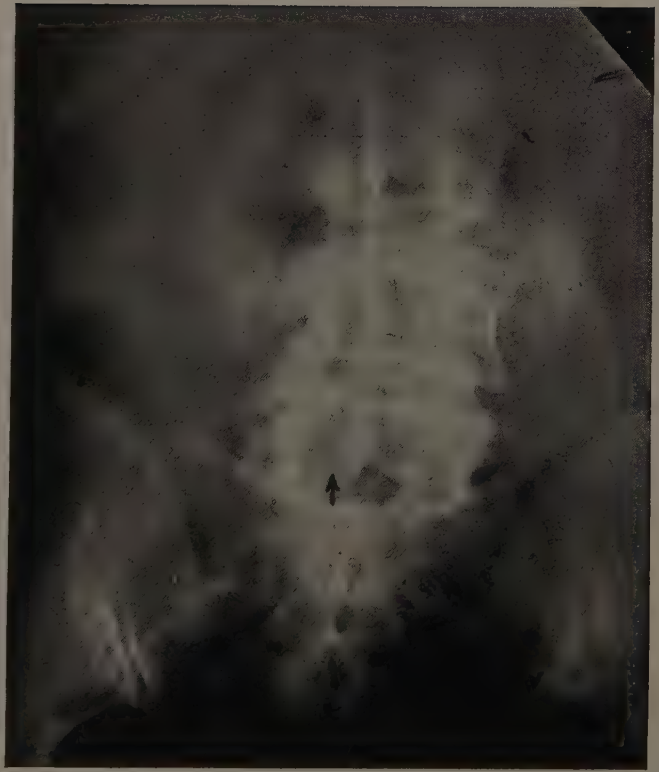


Fig. 52. R.R. No. 46800. Neurofibroma at L 5. Anteroposterior roentgenogram shows atrophy of the laminae (arrow) of L 4 and L 5, particularly on the right side.

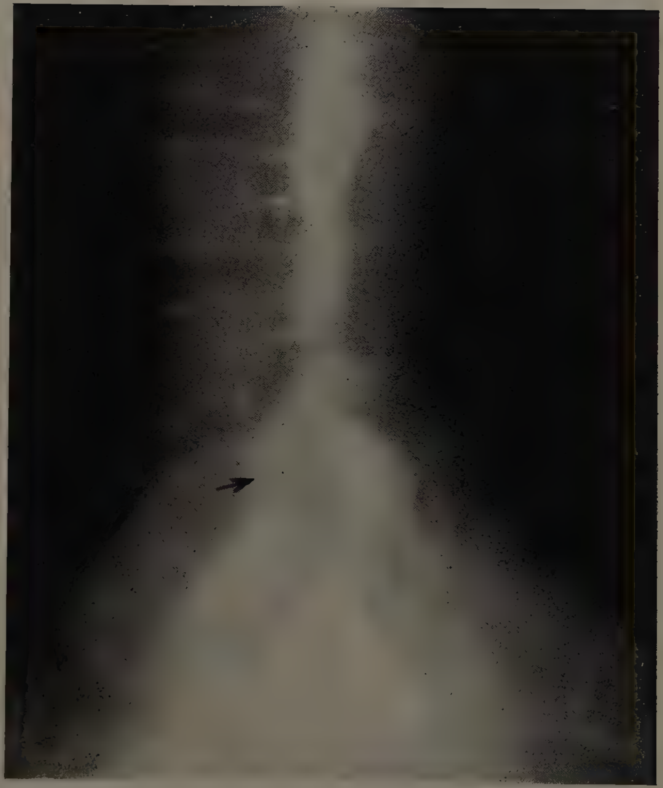


Fig. 53. R.R. No. 46800 (same case as Fig. 52). Lateral roentgenogram shows cavity in dorsal margin of the body of L 5 (arrow).

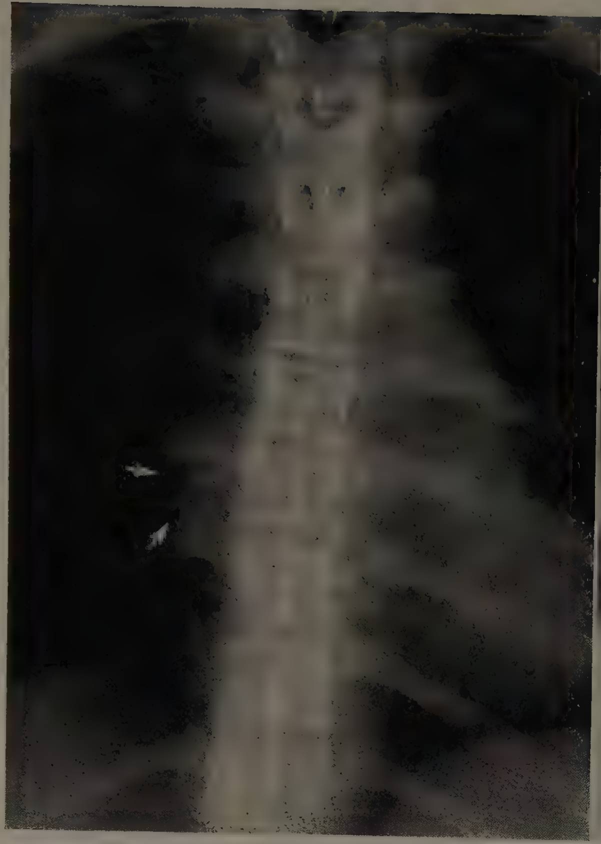


FIG. 54. R.R. No. 34501. Neurofibroma at T 7. The tumor projected through the intervertebral foramen and formed a small soft tissue mass to the right of the spine (arrows).

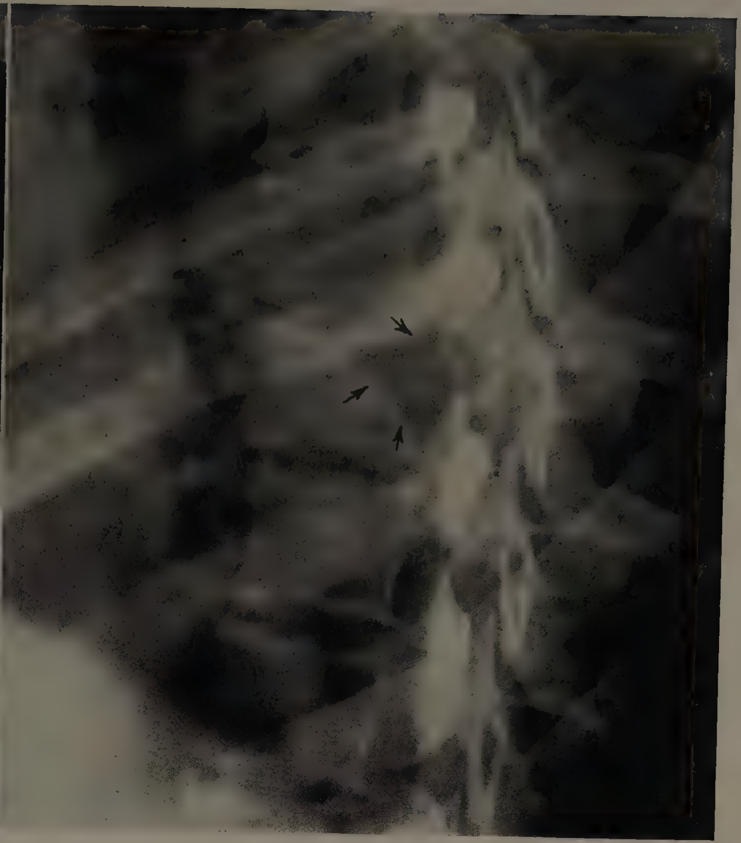


FIG. 55. R.R. No. 34501 (same case as Fig. 54). Oblique view which shows pressure erosion of the right lateral and posterior margins of the body of T 7.

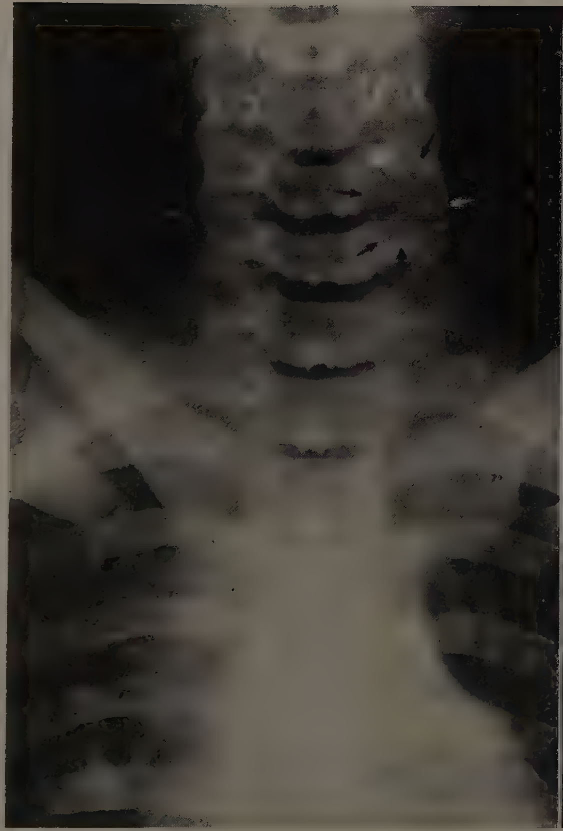


FIG. 56. R.R. No. 29403. Extramedullary neurofibroma involving C 5 and C 6. The pedicles and posterolateral margins of the bodies of C 5 and C 6 are eroded (arrows).

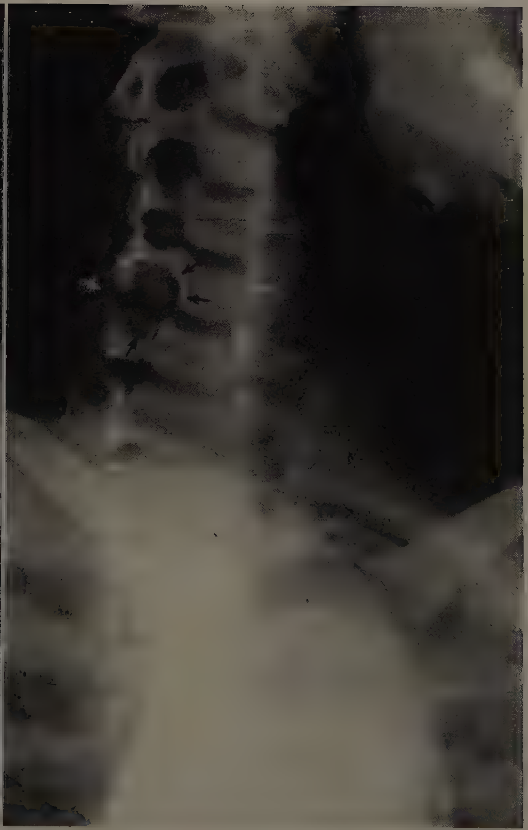


FIG. 57. R.R. No. 29403 (same case as Fig. 56). Oblique view showing the enlargement of the intervertebral foramen between C 5 and C 6 (arrows).

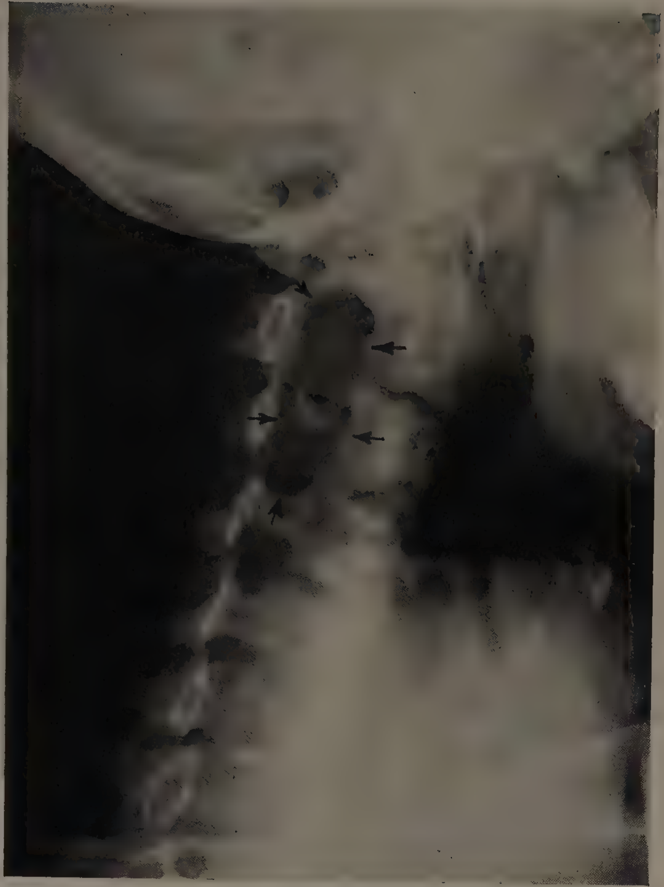


Fig. 58. R.R. No. 32837. Neurofibroma at C 2 and C 3. Oblique view showing erosion of the right pedicles of C 2 and C 3 (arrows).

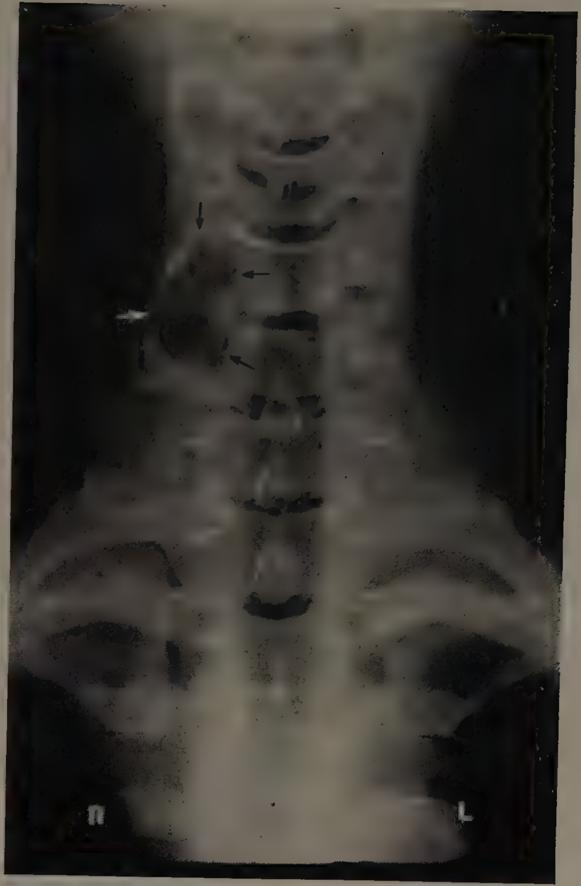


Fig. 59. R.R. No. 17841. Neurofibroma involving C 5 and C 6. Anteroposterior roentgenogram showing erosion of the pedicles and lateral masses on the right side of C 5 and C 6.

ence of large growths, one or both pedicles of a single vertebra may be destroyed. The laminae often are thinned by the pressure of the mass (Figs. 52, 53). The spinous process and the superior and inferior articular processes are also frequently eroded. In the lateral roentgenograms, a concave defect in the posterior and lateral portions of the body of a vertebra is commonly seen (Figs. 54, 55).

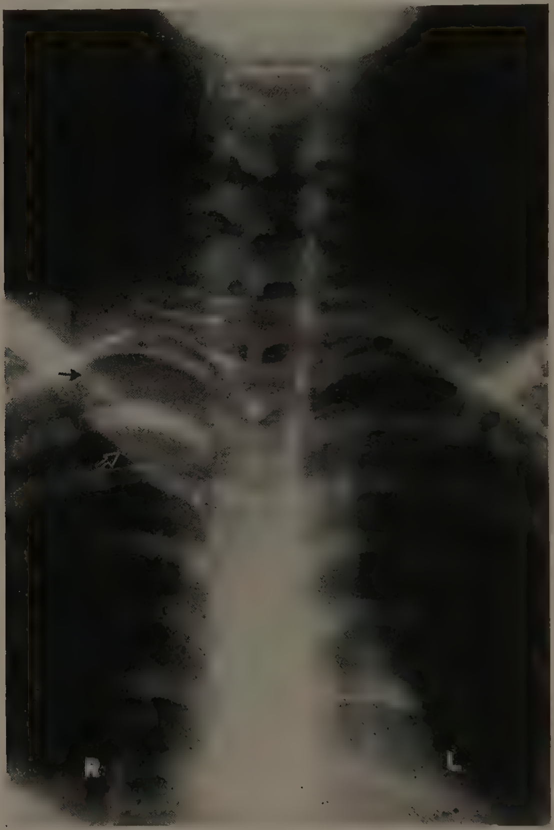


FIG. 60. R.R. No. 5721. Neurofibroma involving the right side of the second and third dorsal vertebrae. Note the large soft tissue mass in the right upper lung field (arrows); and the erosion of the right pedicles of T 2 and T 3. The heads and necks of the right second and third ribs are also eroded, and the intercostal space is increased in width.

Since many of the perineurial fibroblastomas grow through an intervertebral foramen, it is common for this opening to be enlarged due to the absorption of those portions of the pedicles that bound it (Figs. 56, 57, 58, 59). In order to visualize enlargement of the intervertebral foramina, it is necessary to have oblique roentgenograms. The films should be made with the body rotated 45 degrees from the horizontal.

The tumors that extend outside of the spinal column may affect the posterior ends of one or two ribs. Thus the head and neck of one or two ribs may be quite deformed. Occasionally the ribs are separated sufficiently so that the intercostal space on the affected side is definitely greater than on the opposite side.

Another valuable diagnostic sign commonly produced by the neurofibroma, when it extends through the dura and beyond the vertebral canal, is the distortion of the paraspinal soft tissues. The displacement varies from a small bulge, which is barely recognizable, to a mass extending 6 to 8 cm. lateral to the spine. Such distortion of the paraspinal soft tissues is particularly well shown when the tumor grows into the chest cavity where the surrounding lung forms a distinct contrast to the mass (Fig. 60). In a patient with spinal cord symptoms, unilateral erosion of a part of one or two vertebrae, associated with a rounded soft tissue mass adjacent to the spine, is characteristic of a neurofibroma. However, the differential diagnosis must be made between this form of tumor and the tumors arising in the medial and superior portion of the mediastinum or apex of the lung. In these, there often is infiltration and destruction of one or more ribs and the outline of the mass is much less distinct.

These neurofibromas frequently produce an increase in the interpediculate measurements of one or two vertebral segments. The distance is extended beyond the normal in 65 per cent of the cases. In many of these, the enlargement of the canal is only 2 to 4 mm. and is limited to one vertebra, although the measurement may be increased in two segments, as already stated.

Sarcoma. True sarcoma arising within the meninges are infrequent. They have a tendency to grow through the intervertebral spaces and form soft-tissue masses along the spine. The masses produced by the sarcomas are long and flat, unlike the more circular types formed by the neurofibromas. Furthermore, the sarcomas often produce some erosion of the vertebrae. It may be said, therefore, that whenever a spindle-shaped soft tissue mass is seen adjacent to the spine with or without evidence of bone destruction, the lesion is probably a sarcoma (Fig. 61).

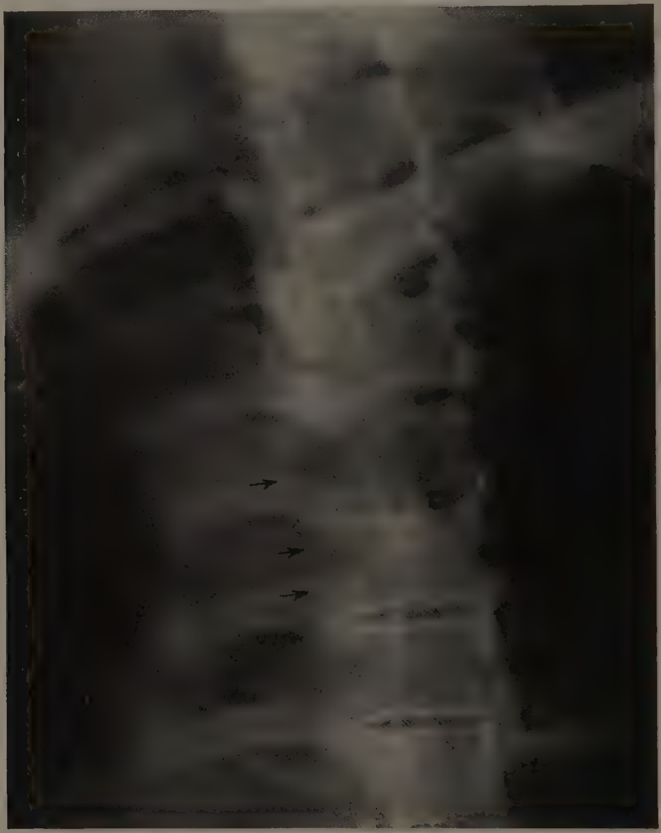


FIG. 61. R.R. No. 41959. Sarcoma extending ventral to T 4 and T 5, best shown in the oblique roentgenograms (arrows).

Varices. Developmental enlargements of the veins within the vertebral canal are common. In some of the cases there is erosion of the pedicles, laminae, and the articular processes, while in others there is no change. The pedicles may show the effects of pressure atrophy in that they are at times concave or flat. The interpediculate measurements are increased in some of the cases, and the enlargement of 2 to 3 mm. commonly involves only one vertebral segment.

Occasionally there is associated with the varices of the spinal canal a congenital malformation of a part of a vertebra (Fig. 62). Thus, in one case the lower half of the pedicle of T 12 was deformed, and in a second case the cortex on the mesial margin of the involved

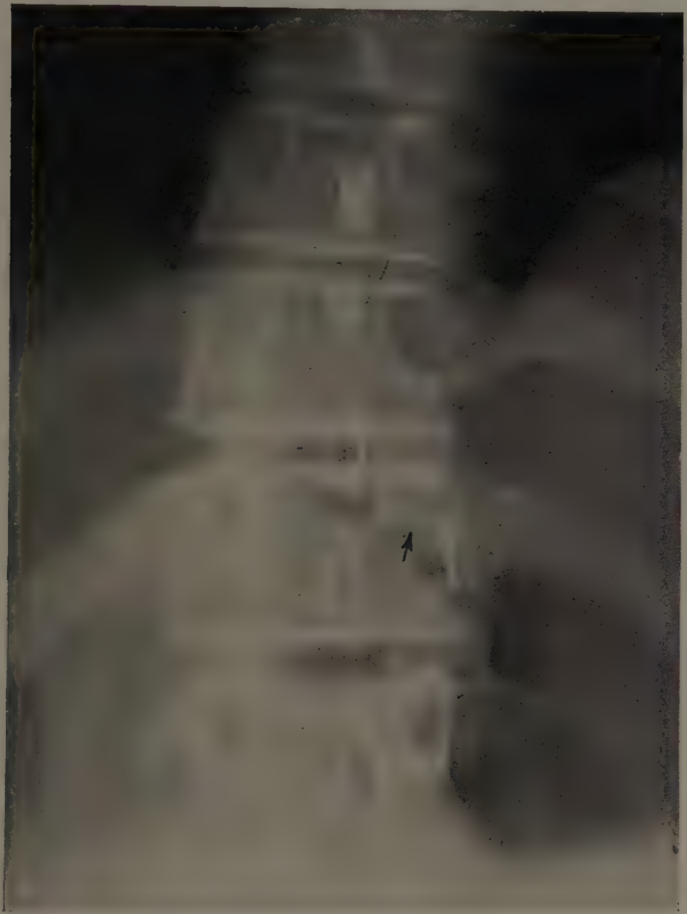


FIG. 62. R.R. No. 24947. Varices. Anteroposterior view shows maldevelopment of the lower half of the left pedicle of T 12 (arrow).

pedicles was extremely thick, so that the medullary portion was practically obliterated.

Giant Tumor of Cauda Equina (page 422). The giant tumor of the cauda equina is one of the few spinal neoplasms which causes atrophy of a number of vertebrae. In fact, one of the types of tumor which falls into the group of giant tumors of the cauda equina is the ependymal cell glioma arising in the filum terminale or conus medullaris (Figs. 38, 39, 40, 41, 42, 43).

Most of the giant tumors of the cauda equina are undoubtedly

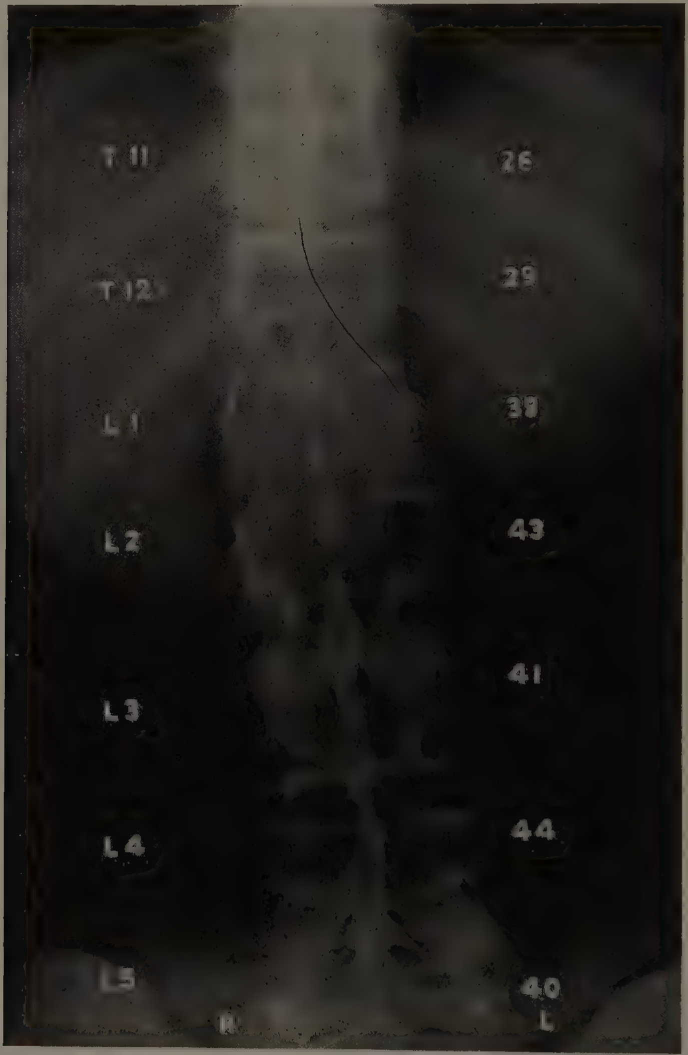


FIG. 63. R.R. No. 19459. Neurofibroma. Anteroposterior view shows marked pressure erosion of the pedicles, laminae, and bodies from T 11 to L 4, inclusive.

ependymomas. In certain instances, a perineurial fibroblastoma may attain an unusually large size and produce changes in the pedicles, bodies, and laminae of a number of the vertebrae in the lumbar region similar to those produced by an ependymoma (Figs. 63, 64).

In the well advanced case, the narrowing and flattening of the pedicle in the roentgenogram is striking. It is difficult, if not impossible, at some segments, to determine the pedicle. At other segments, the pedicles exist merely as thin streaks of bone with the concavity medially placed. Wherever the interpediculate measurements can be determined, it will be found that they are markedly increased. By

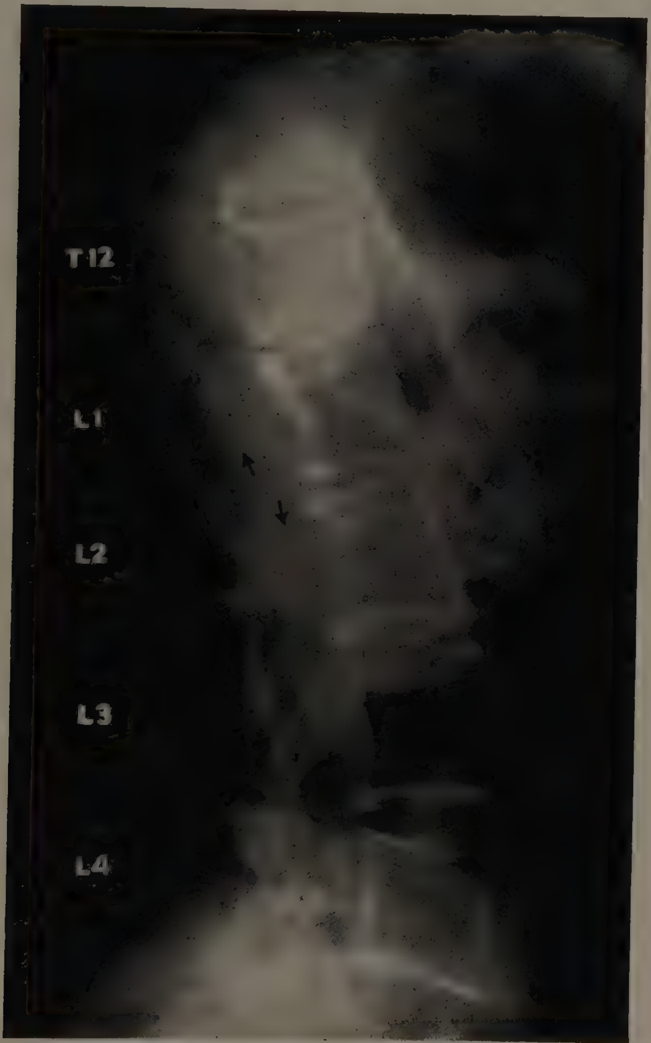


FIG. 64. R.R. No. 19459 (same case as Fig. 63). Lateral view of the lower spine, showing the excavation of the dorsal margins of the vertebral bodies, and the marked thinning of the pedicles of L 1 and L 2 (arrows).

making the measurements, the extent of the tumor can be quite accurately estimated, and this aids in determining the type of neoplasm. The laminae are markedly thin, and in the lateral roentgenogram, hollowing of the posterior surfaces of the bodies is visible. Moreover, in the lateral roentgenogram it is often impossible to recognize the pedicle or portions of the lateral mass of a vertebra.

EXTRADURAL TUMORS (Chapters VII and VIII)

Tumors arising extradurally form about 20 per cent of all intraspinal growths. The extradural tumors cause changes in one or more vertebrae, or in the surrounding soft tissues, which are visible in the roentgenogram in about 80 per cent of the cases. In a high percentage of these, the histologic nature of the lesion can be foretold.

Meningeal Fibroblastoma (*Meningioma*). The extradural meningiomas have the same site of election as the intradural, and cause the same roentgen changes. These tumors infrequently erode the contiguous bone, and even when they do the changes are apt to be slight. The remarks made concerning the intradural meningiomas are equally applicable to those arising extradurally.

Perineurial Fibroblastoma (*Neurofibroma*). The extradural neurofibromas provoke the same changes in the vertebrae and soft tissues as do those arising inside the dura mater. The extradural tumors are more prone to grow through an intervertebral foramen. Because of their similarity in appearance in the roentgenogram to the intradural neurofibromas, the reader is referred to the description of this type of tumor when located in the latter position.

Sarcoma. Extradural sarcomas are common, and although they are somewhat less frequent in the cervical region, they have no predilection for any part of the remainder of the spine. These tumors usually produce a soft tissue mass with consequent bulging of the paraspinal soft tissues. The soft tissue mass may be unilateral or bilateral, and is usually elongated. The paraspinal soft tissues when displaced laterally in the thoracic area are usually distorted over several vertebral segments. The change is a diffuse one, unlike that produced by the neurofibromas. In addition, the sarcomas which arise from paraspinal soft tissues quite often involve the bone.

Hemangioma. A hemangioma generally involves the bone and the meninges simultaneously. The tumor often extends intradurally and may even penetrate the substance of the spinal cord. This type of growth is probably developmental in origin, occurs mostly in the thoracic part of the spine, and commonly involves a single vertebra. It has a characteristic roentgen appearance. There is no true bone destruction, but the affected bone is often less dense than normal, as a result of the thinning of the trabeculae by the dilated cavernous blood spaces. In other cases, the bone trabeculae between the blood-filled spaces are widened and this in turn causes the vertebrae to be denser than normal. Characteristically, once the body of the vertebra is involved, it appears striated in a vertical direction due to the arrangement of the cavernous blood spaces cephalocaudally. Although this vertical striation is considered typical, there is another change visible in the roentgenogram which is equally distinctive. This consists of a spongelike appearance in a part or the whole of the vertebra. The spongelike semblance of the vertebra is due to the "end on" projection of the cavernous blood spaces in the film (Fig. 65). The vertebra concerned may be slightly larger than the adjacent ones, and the pedicles, various processes, and laminae may also show the changes mentioned. A slight displacement of the vertebral fascia and a mild scoliosis opposite the lesion are sometimes noted. A striated or spongy appearing vertebra should lead to the diagnosis of hemangioma.

It is well to observe at this point that the body of a vertebra may be striated in appearance without the patient having any spinal cord symptoms. It seems that the striation of the bone in these cases probably is due to a hemangioma which is limited to the body of a vertebra and does not extend into the spinal canal.

Idiopathic Cysts (Chapter XII). The idiopathic cysts occur in the midthoracic region during adolescence. They are usually extradural, although sometimes intradural, in position. In the past few years, I have observed the roentgenograms of three patients with extradural cysts, and one with an intradural cyst. Their roentgen appearance was so similar that it was possible to make a preoperative diagnosis of the histologic nature of the last two cases. All the patients were

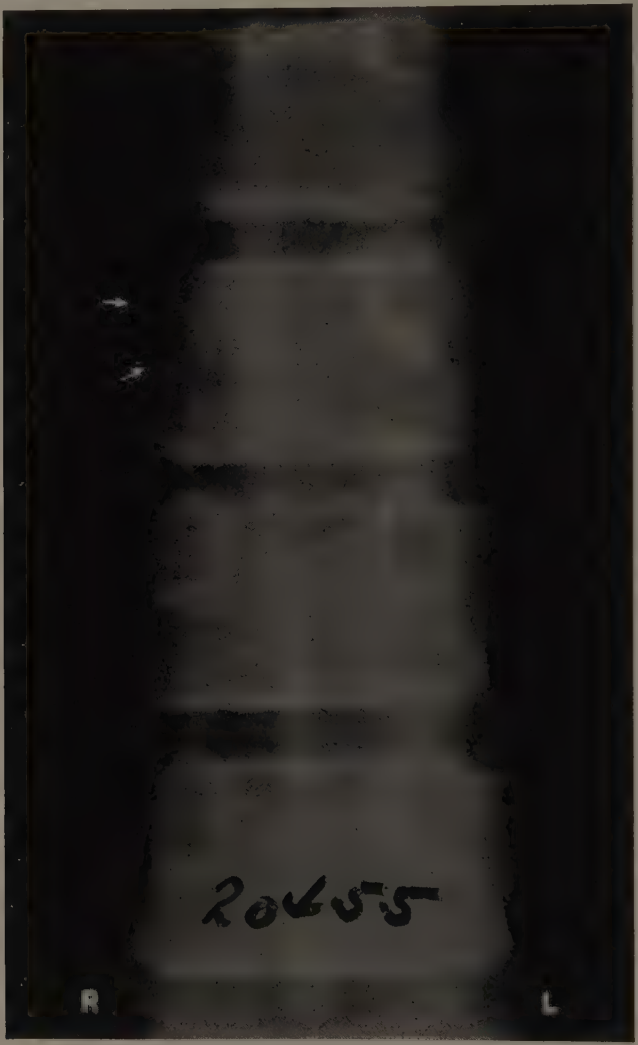


FIG. 65. R.R. No. 20455. Extradural hemangioma. Film shows the cavernous blood spaces which give the vertebrae a spongy appearance and in this case predominantly affect the right pedicle. (arrows).

males. Since the description of these cysts by Elsberg, Dyke and Brewer, numerous other cases have been reported by Lehman, Bucy and Cloward, and others.

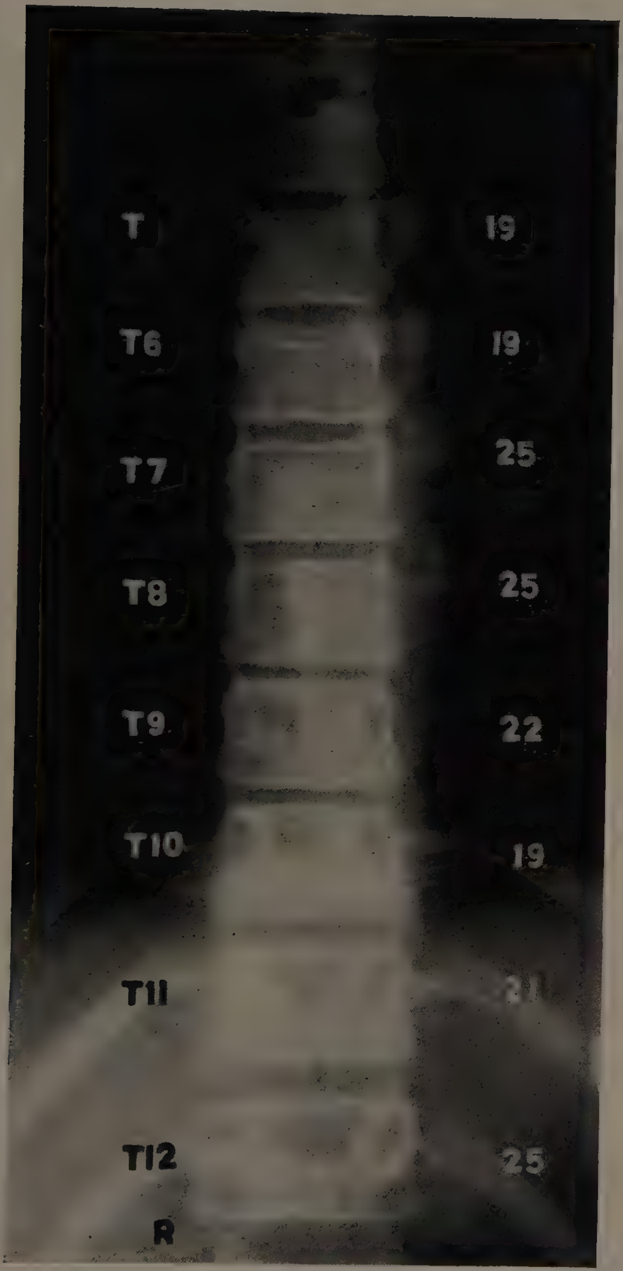


FIG. 66. R.R. No. 25287. Extradural idiopathic cyst. Film shows the flattening of the pedicles of T 7, T 8, and T 9, and the fusiform enlargement of the spinal canal, as indicated by the interpediculate measurements (to the left of the spine).

Bone destruction is the dominant alteration seen in the roentgenogram. This consists of symmetrical absorption of the medial borders of the pedicles of several consecutive vertebrae, so that the pedicles become flat but not concave. The laminae and spinous processes often appear atrophic, and some increase in the normal concavity of the posterior margins of the bodies may take place. The interpediculate measurements are increased over several vertebral segments resulting in a fusiform enlargement of the spinal canal (Fig. 66). The enlargement is gradual and reaches a maximum of about 7 mm. No

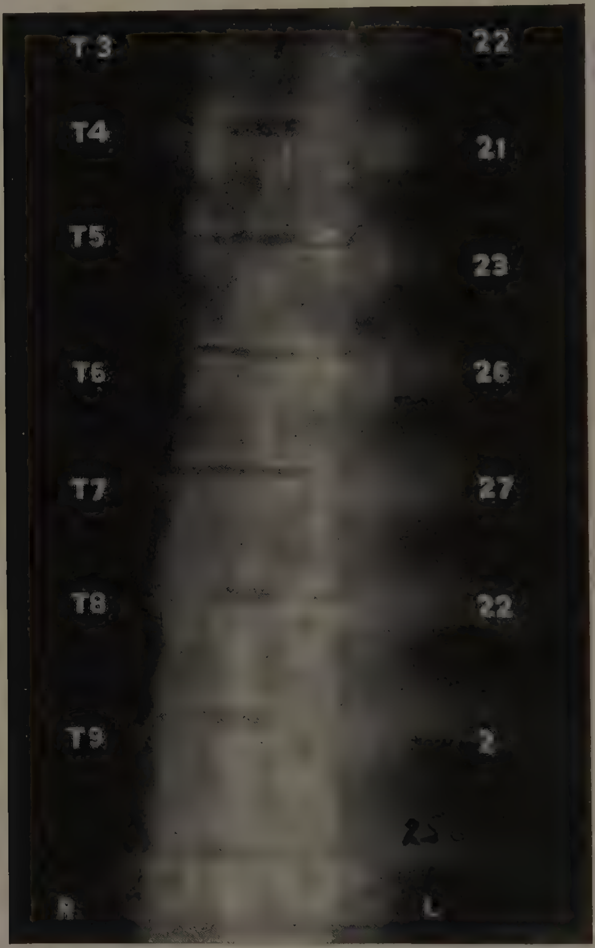


FIG. 67. R.R. No. 25673. Intradural idiopathic cyst. Roentgenogram showing an increase in the interpediculate distance of T 5, T 6, and T 7.

calcification occurs in the cysts, and the intervertebral discs remain intact. The intradural cysts may produce similar changes in the vertebrae as shown in Figure 67. Bucy and Cloward have pointed out that in many of these patients there is an epiphysitis which results in irregularity of the margins of the body of one or more of the affected vertebrae, and the anterior margins of the vertebral bodies may be oblique. These changes undoubtedly are due to pressure of the tumor upon the blood supply to the vertebrae, which results in maldevelopment in the epiphyses and vertebral bodies, known as "kyphosis juvenilis" (Scheuermann's Disease).

Dermoids and Epidermoids (Chapters VII and VIII). The dermoids and epidermoids occur even less frequently within the spinal canal than within the intracranial cavity where they form less than 1 per cent of intracranial tumors.

In the roentgenogram these tumors may produce atrophy of the pedicles and laminae of one or several vertebrae. This results in a localized increase in the interpediculate measurement. No calcification has been described in these tumors. It would seem that at the present time it is impossible to make a diagnosis of this type of lesion, for the changes it produces in the vertebrae are not characteristic and the growths are too rarely encountered.

Parasitic Cysts. In this country parasitic cysts occurring intraspinally are so rare that they are curiosities, and at the present there is no way of making a differential diagnosis roentgenographically.

Ecchondroma (*Ecchondrosis*). Ecchondromas arise from the posterior (dorsal) margins of an intervertebral disc. They are closely allied, if not in some instances identical in origin, with posterior herniations of the nucleus pulposus. However, some undoubtedly are due to proliferation of the dorsal margin of the intervertebral disc. The majority of the ecchondromas occur in males of middle age or over. Most of these lesions are not visible in the roentgenogram, but some of them contain sufficient calcium to be visible. When this happens, a sharply demarcated shadow of increased density, usually about 6 mm. in diameter, is seen in the ventral portion of the spinal canal opposite the level of the intervertebral cartilage (Fig. 68). In a fair percentage of the cases, calcification is noted within the corresponding intervertebral disc, and a slight narrowing of the disc occurs at the site of the lesion. In some cases, moderate generalized osteo-arthritis is present, but this is probably coincidental and of no diagnostic significance. However, the abnormalities described are sufficient for diagnosis.

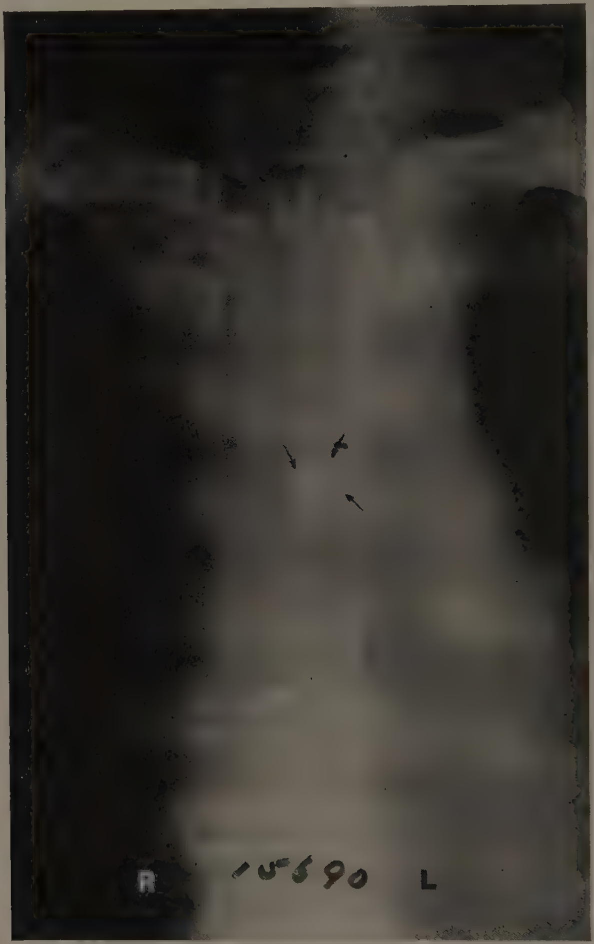


FIG. 68. R.R. No. 15690. Ventral Ecchondroma between T 4 and T 5, showing the partially calcified tumor (arrows).

Lipoma. Bone destruction is the predominating change caused by this type of growth. A number of pedicles usually undergo marked atrophy due to slow and continued pressure (Fig. 69). The pedicles are flat or slightly concave, and the inner borders are frequently unrecognizable in the roentgenograms. The posterior surfaces of a number of bodies may show an increase in their normal concavity, while the heads of the ribs may also be distorted bilaterally.

The interpediculate measurements are definitely increased in a number of vertebrae; in fact, the measurements may be increased in separate portions of the spine and this has not been observed in any other cord tumor.

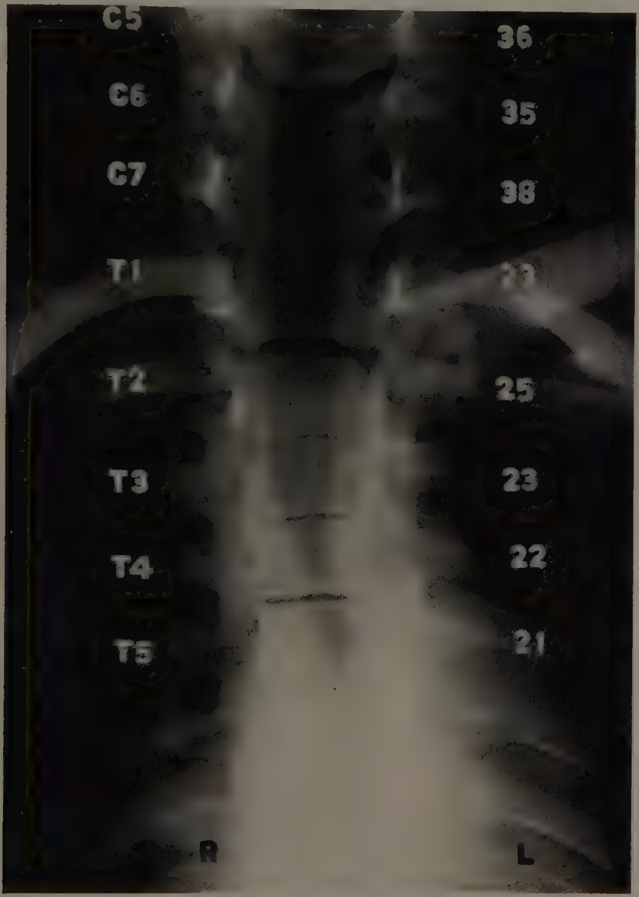


FIG. 69. R.R. No. 14476. Extradural and intradural lipoma. The film (taken after laminectomy) shows a marked increase in the interpediculate measurements from C 5 to T 4, inclusive, with the exception of T 1. A number of the pedicles are either flat or slightly concave medially.

Besides these changes, there may be malformation of the epiphyses of the bodies of several vertebrae, and the bodies themselves often appear square in the anteroposterior views. The intervertebral discs occasionally are narrow, and a scoliosis may coexist.

Lipoma is a rare tumor and whether it occurs extradurally or intradurally, it may produce a considerable enlargement of the spinal canal over a number of vertebral segments.

Neurofibromatosis (Von Recklinghausen's Disease). The typical deformity produced in the osseous structures by these tumors is unilateral erosion of one or two pedicles. The changes in the vertebrae are often slight, while in other cases there is considerable loss of bone. Thus, the lateral masses of two vertebrae may show marked destruction, or the adjacent margins of the ipsolateral pedicles may be affected, causing an enlargement of a single intervertebral foramen.

The interpediculate distance may be increased in one or two segments. Another evidence of neurofibromatosis that I have observed in a single case was a bilateral displacement of the paraspinal soft tissues, so that the apices of both lungs were encroached upon (Fig. 70). This was due to the multiple neurofibromas affecting the brachial plexuses. Furthermore, there was an increase in the width of the retrolaryngeal space, due to the growth of the tumor anterior to the vertebrae (Fig. 71) and behind the larynx and trachea. A kyphosis of the cervical spine also was present. It is to be emphasized that in many patients with neurofibromatosis, no roentgen evidence of the condition may be present.

Chordoma. Destruction of bone is the predominant disturbance produced by this neoplasm and is due to the actual infiltration of the bone by tumor and not to pressure atrophy (Fig. 72). The body of a single vertebra is first affected by the process, which later spreads to the arch. Collapse of the vertebrae results eventually, and destruction of adjacent vertebrae by direct extension of the tumor is common, so that finally several vertebrae are almost entirely destroyed. This involvement of several adjacent vertebrae is frequent with chordoma, and unusual in any other type of malignant disease. The amount of displacement of the paravertebral soft tissues is dependent upon the size of the neoplasm.

In the roentgenogram, when the tumor affects the sacral region primarily, there is pressure erosion of the sacral segments, the number being dependent upon the size of the tumor. In addition, the

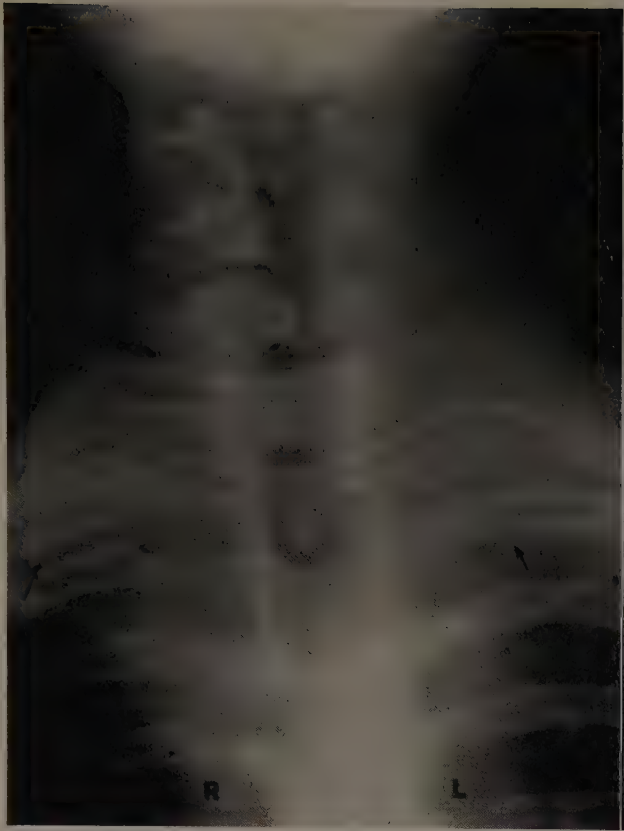


Fig. 70. R.R. No. 22736. Neurofibromatosis. Anteroposterior view showing the encroachment of the tumor masses on the apices of the lungs.

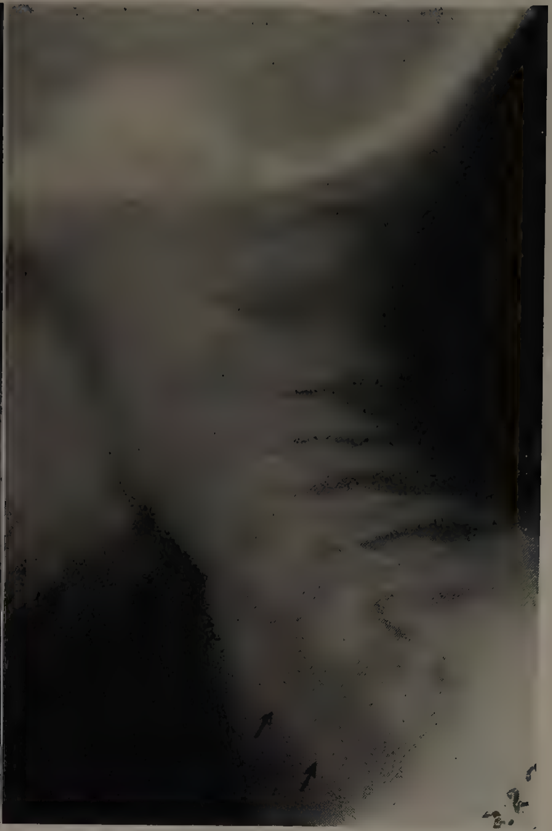


Fig. 71. R.R. No. 22736 (same case as Fig. 70). Neurofibromatosis. Lateral view showing an increase in the width of the retrolaryngeal space (arrows).

tumor may infiltrate the bone and grow into the pelvis. In other cases, it grows posteriorly into the dorsal portion of the sacrum, while in still others, there is definite expansion of the sacrum when

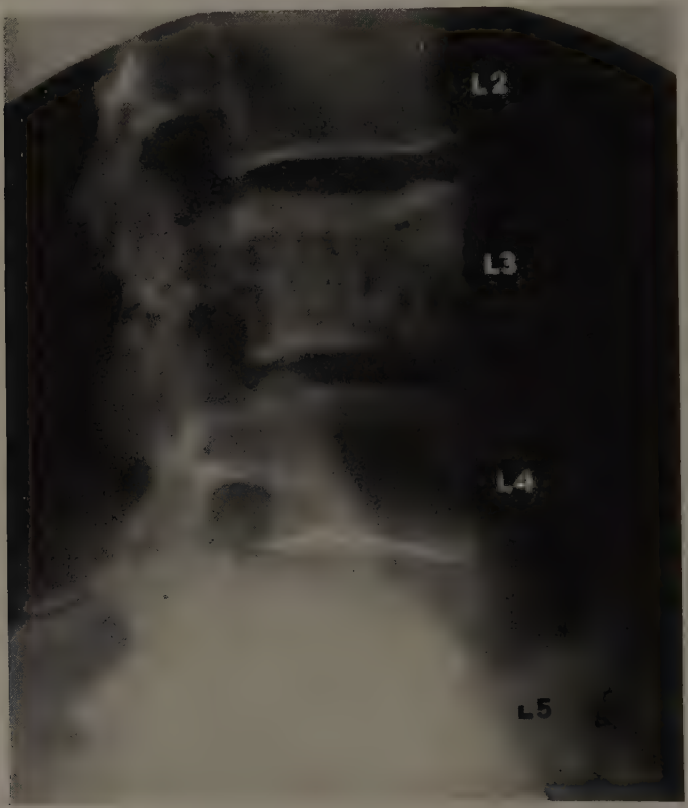


FIG. 72. Chordoma. Lateral view of the lumbar spine, showing partial destruction of the body of L 3. (Courtesy of Dr. George W. Holmes.)

viewed from the side. In the cases in which the tumor grows into the pelvis, a soft tissue mass may be visible in the anteroposterior roentgenograms. The lateral views of the pelvis may also show the mass and in this projection the gas-filled rectum may be displaced anteriorly and the tumor clearly visualized.

Giant-Cell Tumor. A giant-cell tumor usually attacks a single vertebra. Characteristically, the lesion affects the body and also the pedicle, and the growth usually extends into the various other processes. The lesion is primarily destructive, and around the radioluscent areas in the bone there are large, coarse and irregular trabeculae.

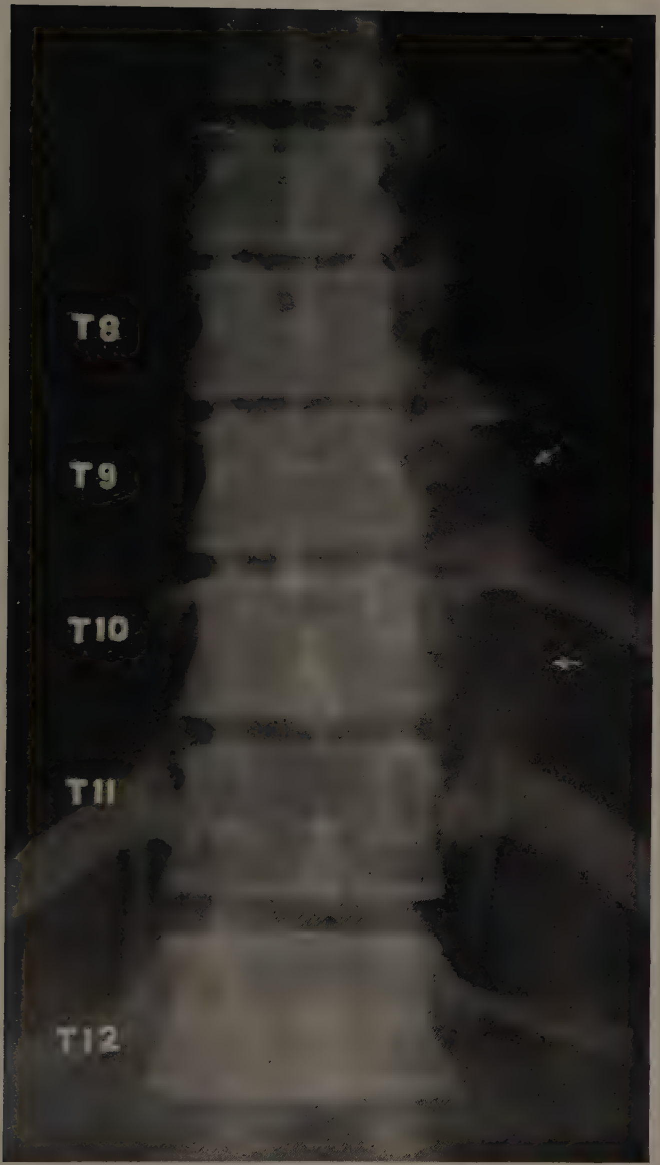


FIG. 73. R.R. No. 26861. Hodgkin's disease producing displacement of the paraspinal soft tissues (arrows) opposite the lower dorsal vertebrae.

Usually the vertebra does not collapse, for the consistency of the growth appears to be sufficient to prevent this complication. A slight localized swelling of the paraspinal soft tissues may be associated with the lesion in the bone.

This type of bone tumor should offer no difficulty in diagnosis or differentiation from a true spinal cord tumor. The only spinal cord tumor that simulates it in any way is the ependymoma, and this form of tumor usually involves a number of vertebrae rather than a single one.

Hodgkin's Disease. Hodgkin's disease affecting the spine is rather uncommon. It produces a localized enlargement of the paraspinal soft tissues and in some cases, soft tissue masses varying in size are seen along the spinal column, either on one or both sides (Fig. 73).

In Hodgkin's disease a hyperplasia of bone with a resulting increase in density of the affected vertebrae may result. If there is some doubt as to the diagnosis, a series of deep roentgen ray treatments should be given to the involved area, and if the mass decreases in size or disappears in several weeks time, the lesion is probably Hodgkin's disease. Usually following radiation, the spinal cord pressure symptoms are promptly relieved, but only temporarily.

Leukemia. Masses of leukemic tissue may invade the meninges, either intradurally or extradurally in several areas, and may displace the paraspinal soft tissues in several locations. Multiple "bulges" of the paravertebral soft tissues may, therefore, be evidence of this type of disease. No other changes are noted in the roentgenograms.

Metastatic Carcinoma. Metastases from a primary tumor frequently involve the spinal column. The site of the primary lesion is usually the breast or the uterus. However, the primary tumor may be in the lung, prostate, thyroid or kidney. Carcinoma of the prostate commonly produces a hyperplastic bone reaction, while the other tumors as a rule produce destruction. The amount of destruction, of course, is extremely variable, but in most instances it has continued to such a point that there is collapse of one or more vertebral bodies. Even when several vertebrae are involved, they are not adjacent to one another but scattered. It is probably well to emphasize here that primary tumors of the spinal cord and its meninges do not produce sufficient destruction of bone to lead to collapse of a vertebral body. In many of the cases of metastatic carcinoma, there is no collapse of the vertebral body but irregular defects are noted in various portions of the vertebrae. Associated with the destruction of bone, a small soft tissue mass often is visible due to invasion by the tumor of the paraspinal soft tissues. The intervertebral disc is not affected in metastatic carcinoma, and this is an important differential criterion from infection.

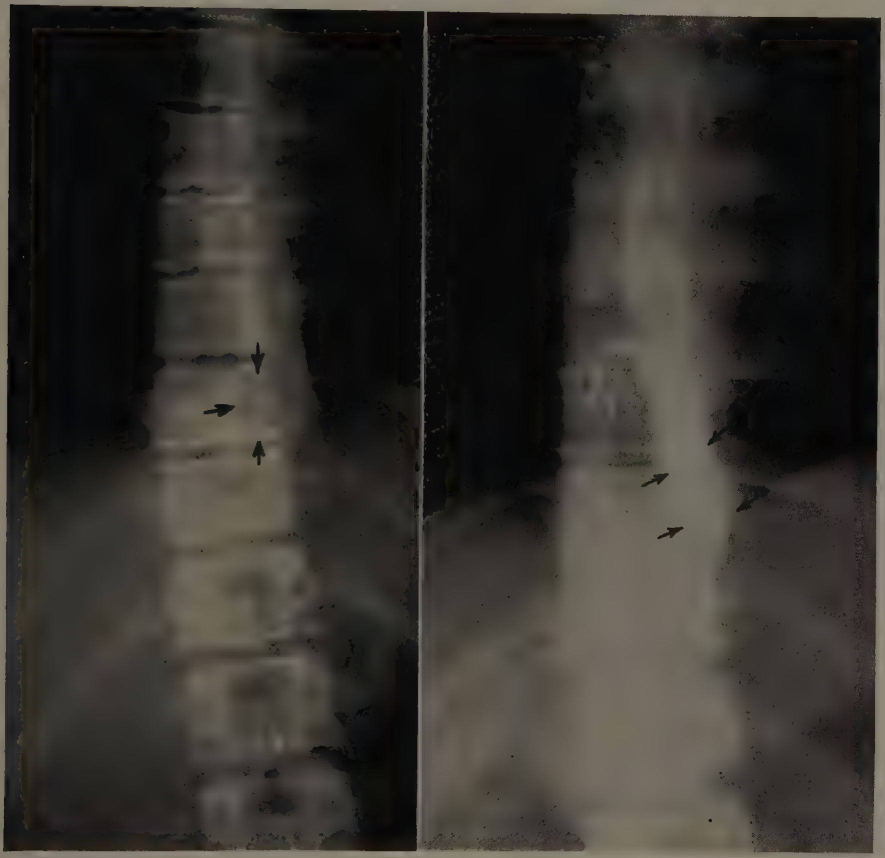


FIG. 74 FIG. 75

FIG. 74. R.R. No. 25976. Osteogenic sarcoma. Anteroposterior view shows destruction of the left pedicle of T 10, and there is a small soft tissue mass adjacent to this vertebra. (Film made Sept. 18, 1933.)

FIG. 75. R.R. No. 25976 (same case as Fig. 74). Anteroposterior view shows considerable new bone formation following radiation. This patient has remained well for six years, and shows no evidence of activity of the tumor. (Examination made Aug. 4, 1939.)

Osteogenic Sarcoma. Osteogenic sarcoma arising in the vertebrae is rare. In one of the few cases which I have observed, there originally was lysis of the left pedicle of T 10 (Fig. 74). Following radiation, this area became dense and there was considerable new bone formation (Fig. 75). The patient has remained well up to the time of writing.

Myeloma. Myeloma arising in a vertebral body is infrequent. In a number of cases, the lesion remains localized in the originally affected vertebral segment and produces advanced destruction of the body of the vertebra which leads to its collapse. These tumors generally occur in patients in middle life or beyond this age period. The upper thoracic portion of the spinal column is the usual site of election in the single form of myeloma.

Lympho-epithelioma (*Schmincke Tumor*). In 1921, Schmincke described a tumor arising in the nasopharynx, which had lymphoid and epithelial characteristics. This tumor is usually quite malignant and its radiosensitivity varies considerably. In rare instances, metastases from the primary tumor develop in a vertebra, and produce a moderate increase in the density of the vertebra.

Osteoma. Benign osteomas of the spine are infrequent. They have a tendency to arise either from the spinous process of a vertebra, or from the lateral mass or from one of its processes. These may reach a considerable size, particularly when they arise in a spinous process.

Roentgenographically, they should give little difficulty in diagnosis. The tumor is sharply circumscribed and has dense cortical bone at the periphery of the irregular extensions. In many of the cases bone trabeculae which have a fairly normal appearance are visible within the substance of the bony mass.

LIPIODOLOGRAPHY

Technique

In 1922, Sicard and Forestier reported the use of lipiodol as an aid in the localization of spinal cord tumors. This method has definite value, but it should be used only when other methods have failed in the diagnosis of the lesion. There are two types of lipiodol:

the heavy, which is heavier than cerebrospinal fluid, and the light, which is lighter than cerebrospinal fluid. Lipiodol is composed of iodine and poppy-seed oil; the amount of iodine determines whether it is heavy or light. Solutions of lipiodol containing varying amounts of iodine can now be obtained. The heavy lipiodol (40 per cent iodine) should be used, since it can be easily controlled and confined to the spinal subarachnoid space. If necessary, the lipiodol may be removed by placing the patient in a vertical sitting position, making a burr hole in the arch of the second sacral segment, and removing the lipiodol by suction. If the light lipiodol is used, a considerable amount may enter the intracranial cavity, and although no reports have been made that the drug has produced injury to the intracranial structures, it seems wise to avoid, if possible, its entrance into the intracranial fluid spaces.

The lipiodol may be introduced either into the spinal subarachnoid space by cisternal puncture or into the lumbar subarachnoid space. With the former method, the patient is usually in the sitting position, while in the latter procedure, the patient is placed on the side with the body well flexed, and a small amount of fluid is withdrawn and the substance introduced. Usually 2 to 5 cc. of cerebrospinal fluid are removed from the spinal subarachnoid space and the lipiodol injected undiluted, as in suspected cases of herniation of the nucleus pulposus; or if a complete block exists, the lipiodol may be emulsified with 2 or 3 cc. of cerebrospinal fluid and then injected into the subarachnoid space. Since the heavy oil is usually employed, the subsequent remarks will allude to its use. If the oil is injected into the cisterna magna, the patient should be placed in the upright position for at least fifteen minutes prior to the roentgen examination. Fluoroscopy may be done immediately following the injection and the opaque substance followed in its course caudally. If the opaque medium is obstructed at any point it is important that the patient be shaken vigorously in order to facilitate, if possible, its downward passage. If the lipiodol is arrested in the lower cervical and upper dorsal region, by the curve which the spinal column takes at this point rather than by a true intraspinal mass, it will be dislodged and proceed rapidly downward. If no obstruction exists, the lipiodol should promptly reach the sacral sac which ends usually opposite the upper margin of the second sacral vertebra. If the medium is arrested higher than the sacral sac, the presence of a tumor is quite certain. Occasionally, the subarachnoid space extends caudally only to the first sacral segment. The appearance of the lipiodol in the caudal end of the subarachnoid sac is like that of a bullet with the "nose" pointing caudally. Sometimes there is a slight bilateral constriction 1 or 2 cm. cephalad to the end of the lipiodol.

A set of stereoscopic anteroposterior roentgenograms and single lateral views of the entire spine should be made. The roentgenograms should be made with the patient in the upright position and in this order, from the cephalad portion of the spine caudally. Moreover, if the lipiodol appears arrested in the first set of films, it is advisable to repeat the procedure the following day to determine whether the block still exists.

Fluoroscopy may be employed immediately after the oil has been injected to determine the presence or absence of a block in the subarachnoid space. If a fluoroscopic table which can be tilted 90 degrees in either direction is available, the patient should be fluoroscoped while he is being tilted up and down, and the progress of the oil noted as it glides through the subarachnoid space.

If the lipiodol is injected into the lumbar subarachnoid space and a tumor is suspected cephalad to the point of puncture, it is necessary to invert the patient so that the head is at least 45 degrees below the pelvis. A greater angle is desirable and the roentgenograms should be made with the patient in this position.

INTERPRETATION

The object, of course, in introducing the lipiodol is to determine whether an obstruction to the free flow of the oil exists, at what level it takes place, and what contours the collection of oil assumes. A complete obstruction to the iodized oil is strongly suggestive of a single spinal cord tumor, but this can also be produced by arachnoiditis. However, the presence of a complete block and a filling defect on the inferior margin of the oil is practically pathognomonic of a spinal cord tumor. All the lipiodol should collect at one level for a complete block. A filling defect in the inferior margin of the lipiodol has the same significance as a filling defect in a barium-filled stomach; namely, it indicates the presence of a mass.

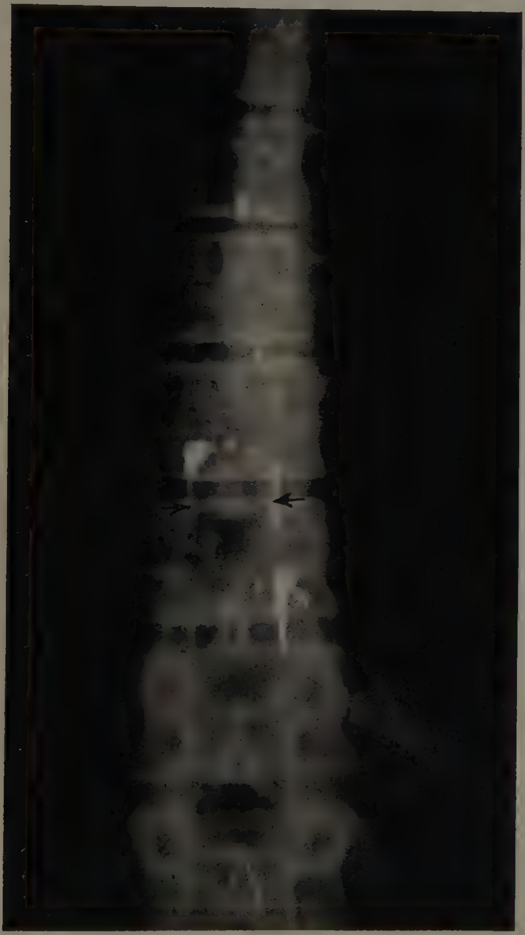


FIG. 76. R.R. No. 31452. Intramedullary tumor. Anteroposterior view made with the patient erect, shows the "capping" of the tumor by the lipiodol.

In many instances, it is possible to determine whether the tumor is intramedullary, extramedullary, or extradural. The intramedullary tumors are often "capped" by the lipiodol. This appearance

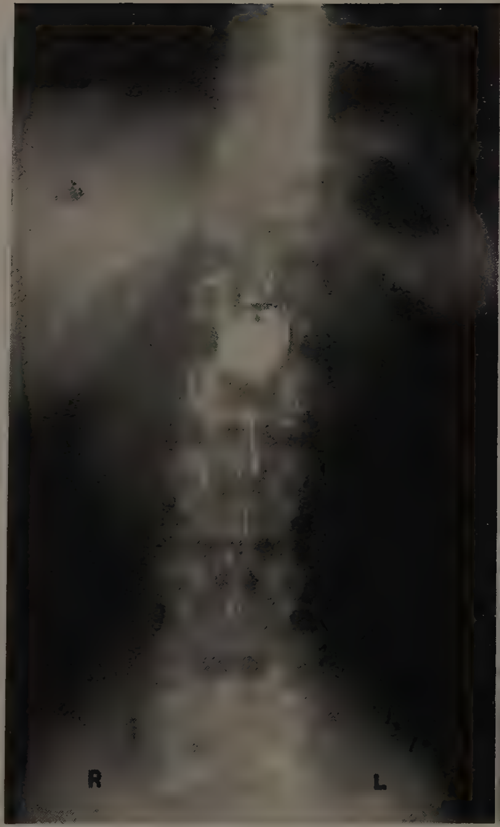


Fig. 77. R.R. No. 16868. Extramedullary neurofibroma. Anteroposterior view of the dorsolumbar region, showing the lipiodol arrested opposite the second lumbar vertebra.

Fig. 78. R.R. No. 16868 (same case as Fig. 77). Lateral view of the dorsolumbar region, showing the filling defect in the inferior margin of the mass of lipiodol.

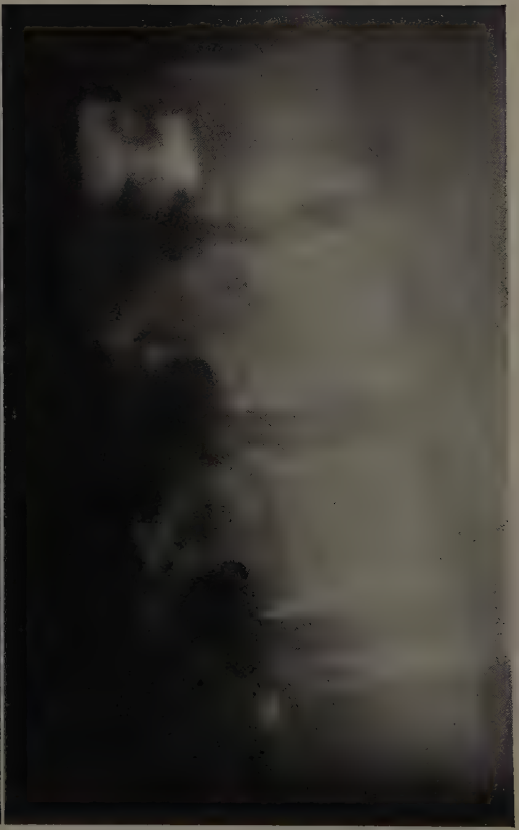


Fig. 79. R.R. No. 23974. Extramedullary neurofibroma. Note the sharp filling defects produced in the lipiodol by the tumor.

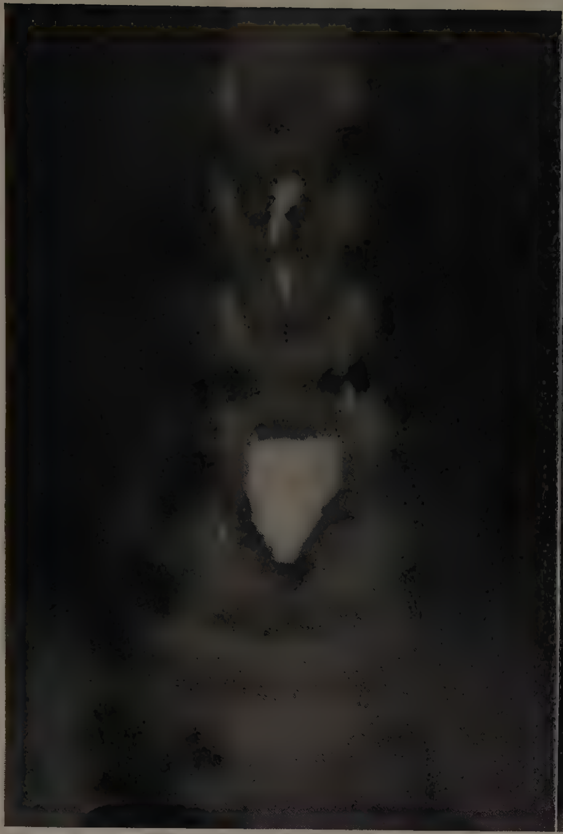


Fig. 80. R.R. No. 31212. Extradural sarcoma. Anteroposterior view showing typical smooth margins to the lipiodol produced by an extradural tumor.

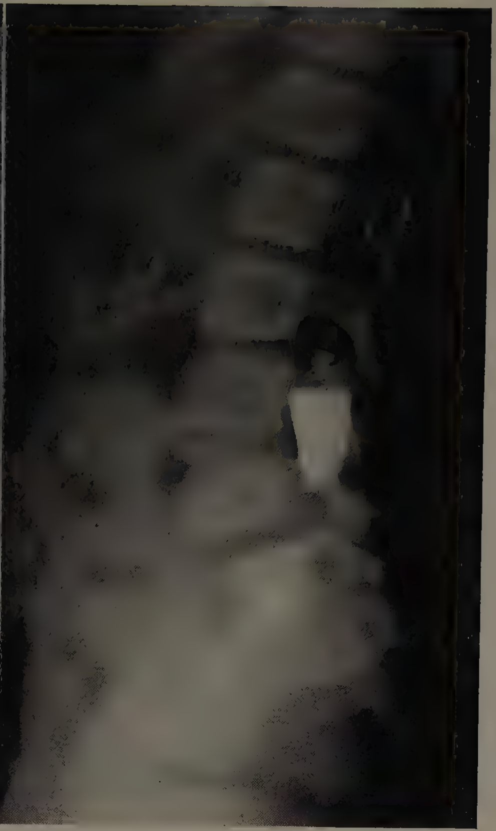


Fig. 81. R.R. No. 31212 (same case as Fig. 80). Lateral view showing the effect of an extradural sarcoma upon the opaque medium.

is due to the fact that the lipiodol attempts to pass along the enlargement of the spinal cord (Fig. 76). On the other hand, the extramedullary tumors produce a sharp filling defect in the inferior margin of the oil. This is because the tumor and the lipiodol are in contact with each other in the subarachnoid space. Therefore, any little irregularity in the margin of the tumor becomes filled with the medium (Figs. 77, 78, 79). In extradural tumors, the margins of the lipiodol are encroached upon in a gradual manner (Figs. 80, 81). This is due to the interposition of the dura between the opaque medium and the tumor.

Incomplete obstruction to the flow of the oil should be interpreted with care, for globules of lipiodol may be arrested by the dentate ligaments, by nerve roots, or by an inflammatory disease of the meninges. In these conditions, no true filling defect is seen in the oil and it is usually scattered in small collections throughout several vertebral segments.

INFECTION

Vertebral Abscess (Chapter vi)

Pyogenic Acute Abscess. Pyogenic paravertebral abscess is uncommon. In the acute abscess, the roentgenograms usually fail to disclose any evidence of spinal disease. The vertebral bodies and arches and the intervertebral discs usually fail to show any evidence of abnormality. In addition, the paraspinal soft tissues are of normal width and contour. In one case seen at the Neurological Institute, there was a marked forward displacement of the trachea at the episternal notch (Fig. 82). In addition, the trachea was slightly narrowed due to the pressure of the mass.

Pyogenic Chronic Abscess. In the chronic pyogenic infection usually no changes are noted in the vertebrae or spinal soft tissues. However, there may be irregular calcification scattered along the dura. In a patient recently examined the calcification extended from the region of T 4 to T 10. The calcification occurred in irregular, small, more or less linear plaques along the dorsal portion of the vertebral arches. It was indistinguishable from the small plaques of calcification which sometimes occur in the meninges and are of no clinical significance.

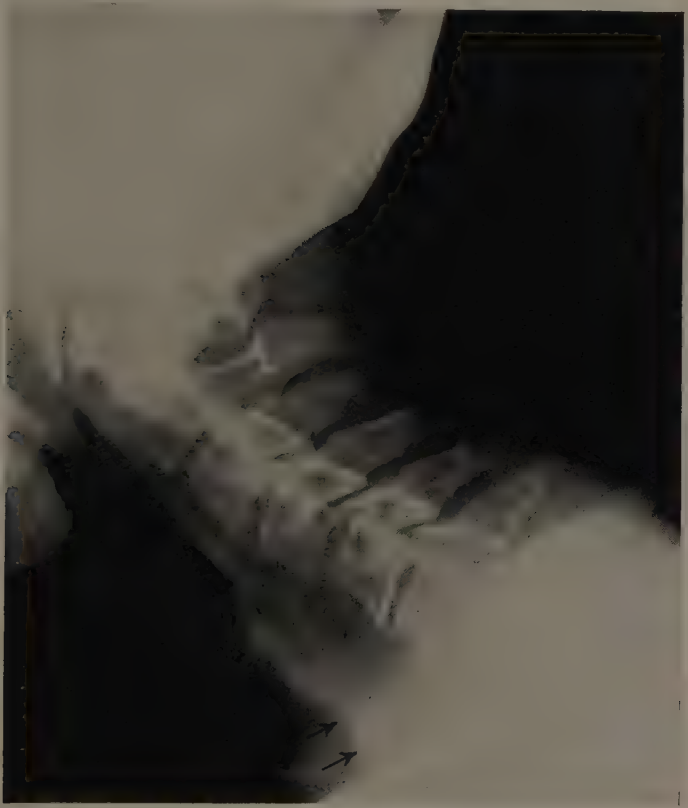


FIG. 82. R.R. No. 25535. Extradural abscess, opposite C 7 and T 1. Lateral roentgenogram showing the anterior displacement and constriction of the trachea at the thoracic inlet.

Tuberculous Abscess. Tuberculosis of the vertebrae is common. This may involve any portion of the spinal column. Roentgenographically, tuberculosis primarily affects the intervertebral disc and later the bodies of the vertebrae. In the roentgenograms, definite narrowing of the intervertebral disc is seen and, in addition, there is usually some destruction of the margin of one or both vertebral bodies adjacent to the intervertebral cartilage. In the advanced cases there may be sufficient destruction of a vertebral body so that collapse occurs. Besides the changes in the bone, there is an increase in the width of the paraspinal soft tissues. This occurs on both sides of the vertebra as a rule, so that a spindle-shaped shadow of soft tissue density is produced. When the lesion is an old one, masses of calcification are seen within the soft tissue mass.

NUTRITIONAL DISEASES OF THE SPINE: XANTHOMATOSIS

Xanthomatosis, a disease of the reticulo-endothelial system which Rowland has done so much to clarify, may affect the spinal cord, meninges, and vertebrae. The deposition of lipid or cholesterin-like substances in any of these structures may be sufficient to produce disturbances in the function of the spinal cord. When the vertebral bodies or arches are involved, a pathologic fracture may occur so that the body becomes compressed and in this way encroaches upon the spinal canal.

Xanthomatosis, when it involves the vertebrae, produces an irregular area of decreased density. Usually, a portion of the margin of the defect is rather dense, but it may be entirely indistinct and irregular without any dense margins. An area of decreased density in the body of a vertebra in a child, should cause one to consider the diagnosis of xanthomatosis.

MISCELLANEOUS

Bone

Paget's Disease (*Osteitis Deformans*). Osteitis deformans, or Paget's disease, frequently affects the vertebrae (Chapter XIII). In Paget's disease, the vertebrae become markedly denser than usual and are enlarged in the anteroposterior and lateral diameters. This refers particularly to the bodies. However, there is also hyperplasia with thickening of the laminae and pedicles of one or several vertebrae. The bodies of the affected vertebrae often are compressed, and the paraspinal soft tissues are increased in width due apparently to osteoid proliferation (Fig. 83). This may be sufficiently well marked to suggest the presence of a true tumor, for it may be localized in one or two vertebral segments. Another common finding in Paget's disease is the marked increase in the normal dorsal curvature.

Osteitis Fibrosa. Osteitis fibrosa cystica is mentioned here, for it occasionally enters into the differential diagnosis of spinal lesions. In the few patients who have had this type of lesion, there was an increase in the density of a single pedicle. The sclerosis of the pedicle may continue so that the cancellous portion is entirely obliterated.

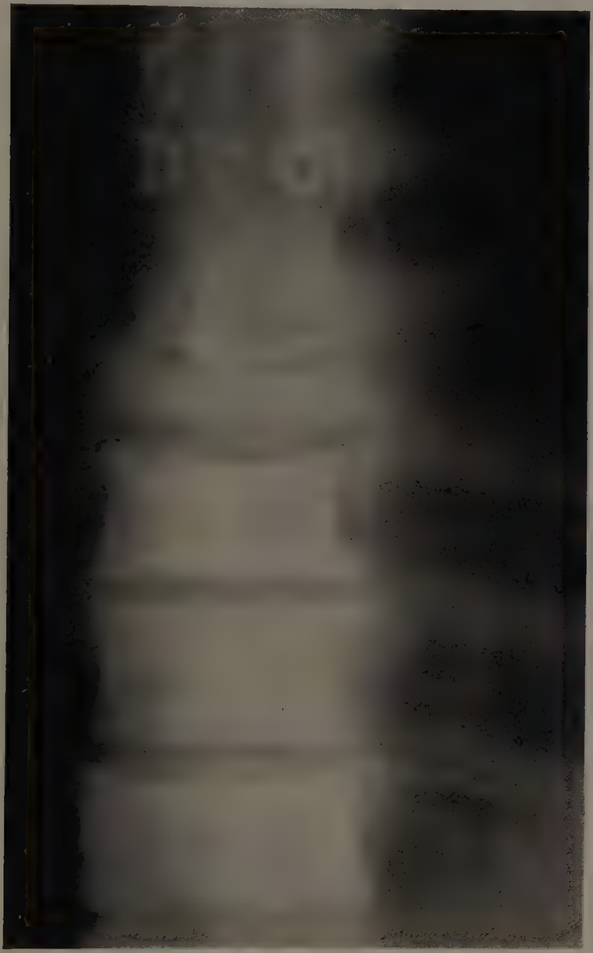


FIG. 83. R.R. No. 45301. Paget's disease. Anteroposterior view showing collapse of the body of T 8; this produced a subarachnoid block and transverse myelitis.

MENINGES

Arachnoiditis. The plain roentgenograms give no evidence of adhesions. After the injection of lipiodol into the spinal canal, complete or incomplete obstruction to the passage of the oil occurs. In some of the cases, the lipiodol may be scattered in small globules over a large portion of the spinal canal. However, there is no characteristic picture of this condition (Fig. 84).

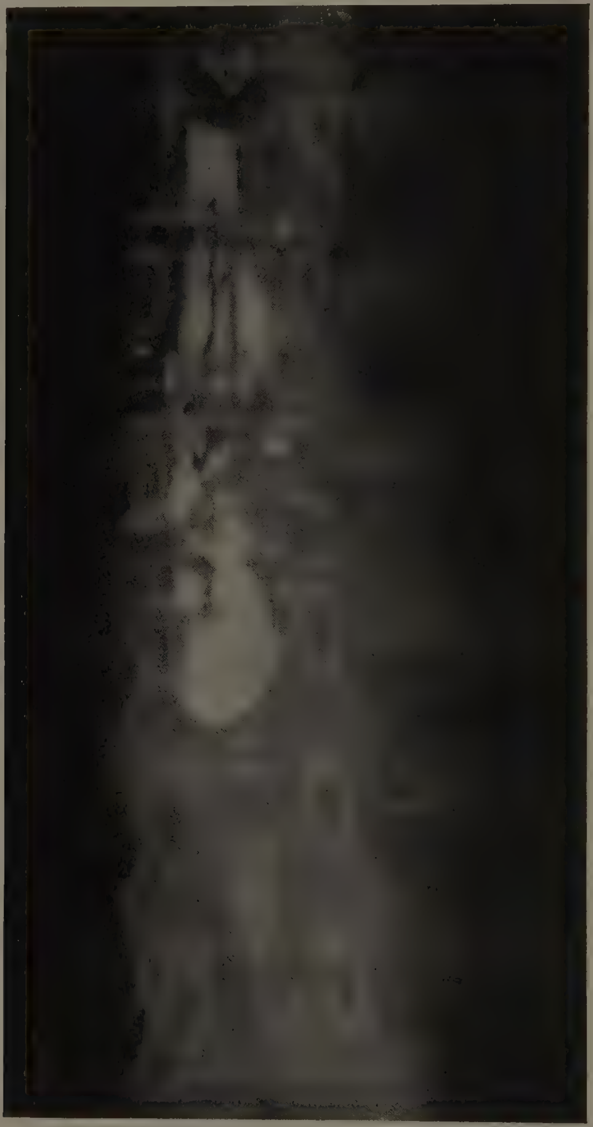


FIG. 84. R.R. No. 41715. Arachnoiditis. Anteroposterior view showing the arrest of the lipiodol at different levels, which is somewhat characteristic of this lesion.

CHAPTER IV

INJURIES OF THE SPINAL CORD AND NERVE ROOTS

THE STRUCTURE OF THE VERTEBRAL COLUMN ALLOWS OF A MAXIMUM OF MOVEMENT WITH A MINIMUM OF CHANGE IN THE relations of the structures within the vertebral canal. The bones are united by strong ligaments, some of which form the actual walls of the vertebral canal (the posterior common ligaments and the ligamenta subflava), while others lie on the outer surfaces of the bones. The entire spine forms a remarkable mechanical system that allows anteroposterior and lateral bending of the column and a certain amount of rotation on its axis. When, however, this remarkable mechanical system is subjected to trauma of sufficient degree, the very factors of strength may become features of weakness.

Sometimes the injury to the spinal cord and nerve roots is caused by trauma to the spine without gross injury to the bony or ligamentous structures; that is, the force produces a sudden dislocation of a vertebra with immediate return to normal of the relations of the bones to each other. More often some of the ligaments are torn, or parts of the vertebrae are fractured. In dislocations in which a spontaneous reduction does not occur, and in fractures with dislocation of fragments, there is always deformation of the vertebral canal and more or less compression of the spinal cord. In most instances the actual degree of spinal cord injury is much greater than the dislocated bone would indicate.

In the cervical region the vertebral canal is relatively large and the vertebrae are more freely movable upon each other so that injuries to the spinal cord without gross bone lesions are relatively frequent; while in the lower thoracic and lumbar regions, where the vertebrae are less movable, injuries to the spinal cord and nerve roots are more often associated with gross bony changes. In the cervical region, on account of the relative disparity between the size of the spinal cord and the size of the vertebral canal, recent dislocation of the body of one vertebra upon another with persistent deformation of the canal is often met with in individuals in whom there are few and slight signs of a lesion of the spinal cord or nerve roots. However, the prominence of the posterior surface of a dislocated vertebral body may project into the canal to such a degree that, secondarily, the spinal cord is subjected to frequent small traumatisms with slowly progressive pathological changes in that structure at the level of the bony deformity. The same course of events may occur in other parts of the spine.

Injuries to the bones and ligaments of the spine are either due to direct or indirect violence. The nature and location of the osseous and ligamentous lesions are dependent upon the location, direction and intensity of the force, and the position of the vertebrae at the time of the trauma. Direct injuries are due to blows upon the back and to direct trauma as from a heavy body striking the spine, "run over" accidents, and the like. Injuries due to indirect violence are more frequent than those due to direct violence. At birth, injuries may occur as the result of manipulations incident to breech presentations. In adult life, they are most often due to falls and diving accidents in which one or more vertebrae are crushed by the force transmitted through the axis of the spine or flexion of any part of the vertebral column, such as occur so frequently in automobile accidents and after direct trauma to the back.

The bodies of the vertebrae may be fractured as the result of muscular violence. Instances have been published of fractures following the spasms of tetanus. Very recently, Polatin, Friedman, Harris and Horwitz (1939), and others have reported that in 43 per cent of patients who had received metrazol in the treatment of schizophrenia and other psychiatric disorders, fractures of the anterior parts of the bodies of some of the thoracic vertebrae were found after the convulsions produced by that drug. The bone lesions were found most often somewhere between the fourth and the tenth thoracic vertebrae.

The injury to the bones and ligaments as the result of hanging is usually found in the uppermost cervical region, involving the first and especially the second cervical vertebrae, the occipito-atloid ligaments, and those which bind together the upper three cervical vertebrae.

In my experience of about 80 cases, injury to the spinal cord and nerve roots from cervical dislocations or fracture-dislocations was found in over 80 per cent of the cases. When the fact is taken into account that with injury to the uppermost part of the spinal cord and medulla oblongata and with traumatic lesions of the brain and of other parts of the body, death occurs so soon after the accident that the individual is not seen by the clinician, the incidence of spinal cord lesions in vertebral fractures and dislocations must be higher than the figures ordinarily given.

In general, fracture-dislocations of the vertebrae complicated by injury of the spinal cord are met with most often at the fifth, sixth and seventh cervical and the twelfth thoracic and first lumbar vertebrae.

In addition to fractures and dislocations of the vertebrae, spinal cord or root symptoms may occur as the result of injuries to the intervertebral fibrocartilages or to the ligaments. The ligaments that are most often affected are the ligamenta subflava and the capsular ligaments. Many years ago, I reported a case in which severe root pain was produced by the compression of a posterior spinal root by a ruptured and dislocated ligamentum subflavum, and other cases have recently been published by Towne and Reichert (1931) and by Abbott (1936). Ruptures of the anterior or posterior common ligaments occur only with extreme grades of trauma.

The symptoms which result from injuries to the spine may appear at once or after the expiration of shorter or longer periods of time. In the latter instance, the symptoms are caused either by secondary changes in the framework of the spine, such as hypertrophy of bone and thickening of ligaments, or by late lesions of the nervous structures produced either by the primary injury or by the secondary effects of a deformation or narrowing of the vertebral canal (the late effects upon the spinal cord of a projection of fractured or dislocated bone into the vertebral canal, etc.)

ALTERATIONS IN THE RELATIONS BETWEEN SPINAL CORD AND NERVE ROOTS AND THE WALLS OF THE VERTEBRAL CANAL

The relative size of the spinal cord and of the canal in which the cord is contained is such that there is, roughly speaking, a space of about one-half centimeter between the walls of the vertebral canal and the cord. The slight mobility of the spinal cord both from above downward and in anteroposterior and lateral directions is limited by the nerve roots and the slips of the dentate ligament. Furthermore, the spinal cord is surrounded by a buffer of fluid which under normal conditions protects the spinal cord from sudden contact with the dura and the bony walls of the vertebral canal. Due to the differences in the course of the spinal roots from the spinal cord to the dura (page 3), the cervical and to a lesser degree the dorsal roots are more apt to be injured when the force which impinges upon the spine is more in the direction of the axis of the vertebral column, while the lumbar and sacral roots are more easily traumatized by a force which is exerted transversely.

When a force which impinges upon the vertebral column is sufficiently great, a temporary change in the shape of the spinal canal is produced. If the direction of the force is in the axis of the column, the column is momentarily shortened or lengthened; if the force is from the side or from the back, the affected part of the canal is momentarily narrowed. The bones may return to their normal relationship without gross injury, but the momentary deformation of the vertebral canal may have caused a more or less profound injury to the spinal cord, varying from a slight contusion to a complete transverse division. On the other hand, if, after the injury, the bones do not return to their normal relationship, or if fragments remain dislocated, the injury to spinal cord or nerve roots due to the first effect of the trauma is complicated by pressure upon cord or nerve roots by the dislocated bone.

The deformity of the vertebral canal most often follows one of several types:

1. With or without some rotation, there is *a forward dislocation of one vertebra* (and of the column above) upon another, so that the anterior surface or its lower border projects into the vertebral canal. In the slighter degrees of this deformity, there may be no gross evidence of compression of the spinal cord or nerve roots, although lateral roentgenograms show that the canal is definitely narrowed by the projecting bone. In the extreme degrees of this dislocation, the body of one vertebra lies anterior to that of the one below it, with, of course, complete obliteration of the canal and complete transverse crushing of the spinal cord. These extreme degrees of dislocation are more often seen in the lower dorsal and lumbar vertebrae after direct blows of great violence.

In the majority of instances, fractures or dislocations of the first and second cervical vertebrae are fatal injuries, but the odontoid process of C 2 may be fractured at its base and slightly dislocated backwards with little injury to the spinal cord or medulla.

2. *Crushing with resulting triangulation of the body of one vertebra.* The anterior part of the body is usually crushed, so that the vertebrae below the affected one run in an abnormal direction forward or the vertebra above is dislocated forward (Fig. 24). As a result the posterior inferior border of the crushed vertebra projects into and narrows the canal, which is angulated at the affected level. This type of injury may be met with in any part of the spine, but it is most often observed in one of the thoracic vertebrae. Such deformation of the vertebral canal is often found without any spinal cord symptoms, but late symptoms from the angulation may occur. Late radicular symptoms often bring the patient to the neurologist.

3. In *fracture dislocations*, especially of the cervical vertebrae, any part of the arch of one or more vertebrae may be also fissured

or broken and dislocated. More rarely, and usually the result of direct violence, the laminae may be fractured and dislocated so that part of the broken arch projects into and narrows the vertebral canal. The fracture may be confined to one arch or several arches may be involved.

4. Many other types of deformities of the vertebral canal occur. They are dependent upon the extent and degree of vertebral fracture and the amount and direction of the dislocation of the fragments.

LESIONS OF THE SPINAL CORD AND NERVE ROOTS PRODUCED BY TRAUMA

The subject of bullet and stab wounds of the spinal cord will be discussed in a separate section. The following general statements have reference to the lesions found in the spinal cord and nerve roots after trauma with or without fracture or dislocation of the vertebrae.

1. *Hemorrhage into the spinal canal,* either extradural or intradural and subarachnoid, rarely occurs without some injury to the spinal cord. Whatever the mechanism may be which causes a rupture of vessels within the vertebral canal (and it is probable that in many instances in which the trauma has not been excessive, it is the veins which are torn), blood within the membranes is very frequent as the result of injury to the spine. However, the amount of blood is rarely, if ever, sufficient to cause any detectable evidence of compression of the spinal cord, because the pressure in the spinal vessels is not high enough to produce a clinically recognizable effect upon the functions of the cord.

2. *Hemorrhages into the substance of the cord* may be either small and recognizable microscopically, or large.

Although there is still difference of opinion regarding the pathology of spinal concussion, it is certain that trauma to the spine may produce multiple small hemorrhages into the substance of the spinal cord. These hemorrhages are usually found around visible vessels and between nerve fibers, and are usually associated with areas of edema surrounding each affected blood vessel. The hemorrhagic and edematous areas are especially apt to undergo secondary softening. In severe crushing of the spinal cord the capillary hemorrhages are regularly found in the cord tissue near and at some distance from the main cord lesion.

When the bleeding into the spinal cord is larger in amount, there is the well known condition of hematomyelia. The bleeding is most apt to involve the dorsal gray substance and the posterior columns. It may occur at the time of the trauma or there may be a secondary hemorrhage a few hours or several days after the injury; or, in rarer instances, one to several months later (Benda, 1926; Else Cohn, 1931; Foerster, 1936; and others). The bleeding into the cord may involve a small area, or may extend over several or many segments. There may be a secondary myelomalacia or the blood may be gradually absorbed, leaving behind a cystic cavity or any area of gliosis and scar tissue.

3. After more severe trauma to the spinal cord, in addition to the changes described under 1 and 2, there may be *destruction of cord tissue*, which varies from division of nerve tissue with hemorrhage to complete pulpification of the cord at the affected level. The tissues are softened and infiltrated with blood, and there is hemorrhage around the various membranes. Only a part of the spinal cord or its entire thickness may be involved. There is often a hematomyelia which extends for varying distances cephalad and caudad to the primarily affected segment or segments. In the most severe injuries, the cord may be entirely divided.

4. *Injuries to one or more nerve roots* are often associated with the cord lesion, especially in the lowest thoracic and uppermost lumbar vertebrae. More particularly in the cervical region, several roots may be crushed and infiltrated with blood. In the lumbar region, on account of the arrangement of the roots of the cauda equina, a small or a large number or all of the roots of the cauda may be injured.

LESIONS OF THE SPINAL CORD AND NERVE ROOTS OF DIFFERENT DEGREES OF SEVERITY

1. Complete Transverse Lesions of the Spinal Cord

The clinical picture of a complete transverse crushing of the spinal cord is a characteristic one: immediately after the trauma there is a complete loss of motor power, of sensation, and of all reflexes below the level of the lesion, with loss of control of the bladder and bowels. However, there may be pain in areas below the level of the lesion on account of connections through the sympathetic nervous system. The only reflex that may persist is a flexor movement of the large toe upon irritation of the sole of the foot. This flexor reflex of Gordon Holmes is believed to be a pure spinal reflex whose arc lies in the spinal cord below the level of the lesion.

The loss of sensation involves all of the dermatomes supplied by the segment of the cord that is crushed and all of those below this level. However, on careful examination, it is usually demonstrable that pain and temperature loss extends to a higher level than tactile loss. This may be explained by the crossing of pain and temperature fibers which, according to Foerster, is complete in one segment, or it may be due to a hematomyelia which extends above the affected segment. If spinal roots which arise from a higher segment than that actually involved by the trauma are crushed, there may be a radicular diminution or loss of sensation on one or both sides of the body in dermatomes which are related to higher cord segments. This is especially apt to be found in the thoracic region, where the spinal roots run a more oblique course.

While there are considerable individual variations, the more exact limitations of the various dermatomes have been extensively studied by Ottfried Foerster, and recent advances in our knowledge of this subject are due almost entirely to the investigations made by Foerster.

In complete transverse lesions there is, as shown by Sherrington, a marked fatigability of the receptors. With repeated irritation of a spot on the skin with a sharp instrument such as a pin, a flexion reflex may occur immediately; if the procedure is repeated a second time, the reaction is less prompt and less marked; if it is repeated a third time, there may not be any reaction. But as soon as some other receptor is irritated, the first reflex may again appear in full strength.

In fresh complete transverse lesions above the lumbar segments, both voluntary and reflex activity of the bladder is lost; the sphincter is in a state of contraction and there is retention of urine. Secondarily, if the distended bladder is not relieved by catheterization, the contraction of the sphincter is overcome and incontinence or a recurring involuntary emptying of the bladder (ischuria paradoxa) occurs. More or less marked priapism is frequently observed, especially after cervical cord lesions.

Intestinal peristalsis is greatly diminished in recent complete transverse lesions of the spinal cord, and obstinate constipation is almost the rule.

The pilomotor and sweating reactions are profoundly affected in complete transverse cord lesions. The former are controlled by the sympathetic, the latter by both sympathetic and parasympathetic fibers. In the majority of cases of fresh transverse lesions, all pilomotor reactions and all sweating are lost in the areas supplied from centers below the level of the lesion. The loss of pilomotor responses and anhidrosis in the dermatomes below the level of the fresh transverse lesions is, however, soon replaced by an increased reflex irritability of the pilo-erectors and by increased sweating of all dermatomic areas reflexly controlled from below the level of the lesion.

Trophic disturbances in areas below the level are also frequent in complete transverse cord lesions, and the great tendency to decubitus in parts exposed to pressure, such as the sacrum, the outer aspects of the hips, the heels, and so forth, need only be mentioned. Edema of the lower limbs and lower parts of the back is often observed. There is, as yet, no satisfactory explanation for the occurrence of this edema.

MOTOR AND SENSORY DISTURBANCES IN COMPLETE TRANSVERSE LESIONS

Lesions at the Fourth Cervical Segment. If the cord lesion is above the *fourth cervical segment*, power and reflexes and sensation are lost in all four extremities and in the trunk up to the neck. Respiration is of the costal type from paralysis of the diaphragm. However, Oppenheim described a case (and I have observed a similar one) in which good movements of the diaphragm were preserved even after a complete transverse lesion of the spinal cord above the fourth cervical segment. The sensory disturbances involve all areas up to the anterior aspect of the neck (C 3) and perhaps also the superior surface of the shoulders (C 4). Rarely, the loss of sensation extends up to the dermatomic areas for the mandibular division of the trigeminus. Sensory disturbances which extend as high as this are rarely seen in high transverse cervical cord lesions, because there are so often injuries to the medulla and the brain, but they are not rarely observed in incomplete cord lesions at this high level. Irregular fever is frequent in these high cervical lesions and its appearance is usually an ominous sign. It is characteristic of this hyperthermia that the patient may be very comfortable and does not appear to be suffering from temperatures of 104° F. or over.

Lesions at the Fifth Cervical Segment. The diaphragm will still contract, as can the trapezius, sternomastoid, and platysma muscles. The rhomboids and levatores scapulae are relatively slightly affected. Power in the biceps is lost and the biceps reflexes cannot be elicited. The triceps and ulna reflexes are often hyperactive. The sensory disturbances involve all areas up to the clavicles and the outer and upper parts of the arms (C 5).

Lesions at the Sixth Cervical Segment. The shoulder muscles are unaffected but the biceps is paralyzed while the triceps reflex is preserved or hyperactive. According to Foerster, tapping the spine of the scapula or a blow on the outer surface of the flexed forearm will cause an outward rotation of the arm. This increased irritability of the external rotators is due to the paralysis of the internal rotators (C 6 to T 1). The sensory disturbances extend up to the outer aspect of the shoulder and the anterior surface of the arm and forearm, the radial aspect of the hand inclusive of the thumb and index finger and the greater part of the dorsum of the hand (C 6, according to Foerster).

Lesions at the Seventh Cervical Segment. The supraspinatus and infraspinatus, teres minor, deltoid, biceps and supinator brevis are not affected, but the triceps is always paralyzed, as are the muscles of the hands and fingers. There is often inversion of the triceps reflex on one or both sides—percussion of the triceps tendon being followed by a flexor movement at the elbow. This inversion of the triceps has been observed by me when the outer aspect of the lower end of the ulna was struck with the percussion hammer. Foerster has called attention to the fact that, in these patients, tapping of the radial side of the back of the hand is followed by a movement of supination of the hand. Furthermore, a blow on the back of the hand will produce a radial extension of the hand, because of heightened irritability of the extensor carpi radialis. In every case of transverse cord lesion above the first thoracic segment, there is a paralysis of the pupillary dilator and, therefore, miosis. The sensory disturbances involve the trunk and lower limbs and part of the anterior aspects of the arm and forearm, and very often the third, fourth and fifth fingers. In my experience, there is considerable variation in the radicular supply of the fingers: sometimes all of the fingers are supplied by C 7, at other times only the ulnar three fingers, and at still other times there is some supply also from T 1.

It is rare that in complete transverse lesions of the cord, a definite distinction can be made between the seventh and the eighth cervical segments, because both segments are so often involved. However, in some instances I have concluded that the injury was primarily in the eighth cervical segment, because the fingers could be extended (extensor communis digitorum), but real flexion of the fingers was impossible. The presence of the Horner symptom complex (enophthalmos, ptosis, and miosis) is also not rare in lesions at the eighth cervical (and first thoracic) segments.

Lesions at Thoracic Levels. In transverse crushing of the *first thoracic* segment, there is always a Horner syndrome, and the miosis, enophthalmos, and narrowing of the palpebral fissure on one or both sides is often very marked. Power in biceps and triceps is preserved and the biceps and triceps reflexes are normally active. There is usually a marked weakness of finger movements as far as their control by the intrinsic muscles is concerned, but the abductor pollicis brevis is always completely paralyzed (Foerster).

Complete transverse lesions of the second to sixth thoracic segments are characterized only by the level of the sensory and motor disturbances. In lesions of the *sixth thoracic* segment, the upper part of the rectus muscle may still be found to contract when the patient is instructed to strain. Affections of the abdominal organs and pelvis are painless when the lesion is at or above T 6. In lesions of the eighth to tenth thoracic segments, the upper and lower abdominal reflexes are lost, as in lesions above this level; but, in my experience, the upper abdominal reflex may still be weak when the lesion is at the tenth thoracic segment. The upper abdominal reflexes are preserved when the lesion is below T 10. In lesions at the twelfth thoracic segment, the lower abdominal reflexes may be definitely weak, so that it appears as if more of the nerve supply for the lower abdominal reflexes is derived from T 12 than from T 11. In transverse lesions below T 11, the sense of fullness when the bladder is distended is preserved.

Complete lesions at lumbosacral levels are characterized by disturbances not only of the cord segments but also of the roots, which are often crushed together with the cord. It is rare in crushing of the lumbar cord to have only one segment affected. Most often several of the segments are affected together. When the lesion involves the second lumbar segment, the abdominal reflexes are preserved while in the male, the cremasteric reflexes are lost. Foerster states that in the transverse syndrome at L 2, the function of the iliopsoas is undisturbed, while the sartorius is very weak, but in spite of the weakness of the latter muscle, the thigh can be bent almost 90 degrees when the knee is kept flexed. In all transverse lesions between L 1 and S 2, the desire to empty the distended bladder is lost.

In complete crushing of the cord and roots of the cauda equina at the level of the third lumbar segment, the patellar reflexes are lost and the leg cannot be extended at the knee. Good flexion at the knee is possible, however, on account of the activity of the sartorius and gracilis. When the lesion is at the level of L 4, the patellar reflexes are preserved and the ability to extend and flex the leg on the thigh is preserved, although power in the quadriceps extensor femoris is weak. A lesion at L 5 leaves the quadriceps and tibialis anticus and tibialis posticus unaffected.

What has been said of transverse lesions of the lumbar segments is true also for lesions of the sacral cord: in traumatic lesions most of the segments are involved and it is rare to find the lesion (and the signs of disturbed function) limited to one cord segment. The clinical picture of lesions of the sacral segments is really that of the roots of the cauda equina at the level of the highest affected roots.

In lesions of the cauda equina at the level of the sacral roots, the disturbances of the motor power and of sensation can be deduced from a knowledge of the nerve supply to the muscles and to sensory areas, and need not be described in detail. There is still some difference of opinion regarding the relative part played by different sacral roots, but the main facts can be found in the description given by Foerster. Mention must be made, however, that in lesions at S 1 and S 2 there is more or less diminution in the power of muscles which control the movements of the leg and foot, while at S 3 all the muscles of the lower limbs have normal power and the reflexes can be normally elicited. In transverse lesions of the cauda equina at S 3, only the bladder, rectum, and genital organs are involved. There is either retention of urine or incontinence, but the latter is by far the most frequent. There is usually rectal incontinence and complete relaxation of the rectal sphincter.

Partial Lesions of the Spinal Cord

The clinical picture of a lesion which does not involve the entire thickness of the spinal cord, is similar, of course, to that observed in complete lesions, excepting that the functions of all of the structures related to the cord segments below the injured one, are not completely lost. Although various combinations occur, it is clinically useful to divide the cases into the following groups:

1. Spinal hemiplegia or hemiparaplegia
2. Monoplegia of an upper or lower extremity
3. Hematomyelia and lesions primarily of the gray matter
4. Injuries to spinal nerve roots, especially injuries to the roots of the cauda equina.

1. Spinal hemiplegia is rare after trauma of the vertebrae, but is seen in its purest form in bullet and stab wounds of the spinal cord (*q.v.*). In spinal hemiplegia there is a loss of power in the limbs on one side, with sensory disturbances predominantly on the opposite side as far as cutaneous sensation is concerned, and on the same side as regards the deep sensibilities (vibratory sense, articular sense, etc.). The level of the lesion is determined from the highest motor and sensory disturbances. As was described for complete transverse lesions, the exact level may be difficult to determine because of associated hematomyelia. In this connection, also, it is well to take into account that the disturbance of pain and temperature sensibility usually extends to a somewhat higher level than that of touch. In fresh injuries which have caused the clinical picture of spinal hemiplegia, the tendon reflexes on the same side are usually depressed or lost, but this diminution disappears after a few hours. The abdominal and cremasteric reflexes on the same side are also greatly diminished or lost; such a depression of the cutaneous reflexes may persist for a long period.

The signs of a lesion limited to one-half of the cord, that is, a typical Brown-Séquard syndrome, are almost never observed. There is usually some additional injury to the posterior or the lateral column of the other side, with disturbances of cutaneous and deep sensibility on both sides of the body.

2. Paralysis of one upper or lower limb may be found after a recent spinal injury, but it is most often found as the residuum after tetraplegia or paraplegia. A supranuclear monoplegia may be the result of a spinal cord injury in which only a part of the pyramidal pathways has been injured. It is inherently probable that the fibers in the pyramidal pathways are arranged in groups for entire limbs or parts of limbs—those for the lower limbs lying furthest away from the midline and those for the upper limbs most mesially. After injuries to the cervical cord, isolated paralysis of one upper limb is more frequent than paralysis of a lower limb without involvement also of the upper limb.

3. More or less hemorrhage into the substance of the cord occurs in every cord injury. A pure hematomyelia with the characteristic signs of a lesion in which the gray matter is mainly affected may exist as the only evidence of a fresh spinal trauma, or it may occur a number of days after a relatively slight injury to the back. It is not rare that signs of a severe cord injury disappear within a few days or weeks and the clinical syndrome of a hematomyelia remains. Dissociated disturbances of cutaneous sensibility without involvement of the deep sensibilities characterize the lesion. Pain and temperature sensibility are profoundly affected, while the ability to recognize light touch is usually preserved. The deep sensibilities—vibratory and articular senses—are usually unaffected. The dissociated disturbances of cutaneous sensibility may involve the entire body, or be most marked nearest the level of the lesion, or they may be entirely limited to dermatomes at the level of the lesion. The occurrence of diminution or loss of pain and temperature sensibility most marked near the level of the lesion is one of the facts that supports the belief of a lamellation of fibers in the spinothalamic tracts. In many instances, as the result of spinal "shock," the first effect of the intramedullary hemorrhage is to cause a loss of all tendon and cutaneous reflexes below the upper level of the lesion. As soon as the primary effect has subsided, the motor disability is

mainly nuclear from involvement of anterior horn cells, and the tendon reflexes of the affected muscles are regularly lost. If the hemorrhage is of sufficient size to make pressure upon the descending pyramidal pathways, there may be left a paresis with spasticity and increase of tendon reflexes of the lower extremities. Muscular fibrillations are rarely observed in fresh hematomyelia, but they appear after the expiration of a few days or weeks when the primary shock effect of the hemorrhage has subsided.

Loss of control of the bladder and rectum occur almost regularly, and vesical and rectal troubles are often a residual symptom when most of the other disturbances have disappeared. The supranuclear pathways for the bladder are evidently diffusely spread over the transverse diameter of the spinal cord, but probably they are situated mainly near the gray matter. When only one-half of the cord is affected, there are often no bladder disturbances.

4. Lesions of the spinal nerve roots. In the majority of instances of trauma to the spinal cord, there is also a lesion of spinal nerve roots. More rarely, roots are injured without any evidence of cord lesion. Below the tip of the conus, however, isolated injuries of the roots of the cauda equina are a frequent result of fracture-dislocations of the lumbar vertebrae. The effect of a slight degree of trauma to posterior roots may be limited to irritation with resulting pain which is projected into the dermatomic area partly supplied by the root. Not only may there be spontaneous pain but also a hyperesthesia or hyperalgesia in the dermatomes that are affected. If the injury involves thoracic posterior roots, there may be abdominal pain and typical visceral crises. I have seen a patient with a fracture-dislocation of the eighth and ninth thoracic vertebrae, in whom the visceral crises were so severe that an acute condition within the abdomen was at first suspected.

If the trauma to the posterior roots has been severe enough to cause complete crushing of one root, ordinary sensory tests may not give any evidence of diminution of sensation on account of the overlapping of dermatomes, but very careful examination with tests of fatigability will show that there is a very slight degree of disturbance. If several roots have been divided, there is always a sensory disturbance which involves pain and temperature more than tactile sensibility. The sensory level of a cord lesion may extend higher on account of an area of radicular loss.

Isolated injuries to the anterior roots are of rare occurrence. The disturbances will be referred to the muscles supplied by them (atrophy, fibrillation, loss of reflex, electrical reactions of degeneration).

Injuries to the Roots of the Cauda Equina

The lower end of the spinal cord terminates at the lower border of the first lumbar vertebra. If the bony injury is lower than this, a neural lesion may be limited to the roots of the cauda equina. If the spinal trauma has been a severe one, all of the roots of the cauda equina below the level of the vertebral lesion are affected; in lesser degrees of injury, only some of the roots or those of one side are affected. The extent of the lesion and the number of roots involved can be determined from studies of the sensory and motor disturbances found on examination. If the bone injury involved the second lumbar vertebra, there is usually some lesion of the conus.

The signs of complete lesions of the roots of the cauda equina have already been described (page 135), but it is evident that the lower the injury, the fewer are the pairs of roots that may be injured and the more limited is the flaccid paralysis due to trauma to the anterior roots and the disturbances of sensation due to involvement of the posterior roots.

If the injury is below the point of emergence of the second lumbar roots, the sensory disturbances will involve dermatomes as high as the middle of the thigh anteriorly and the buttocks posteriorly. With involvement of the fourth lumbar nerves, there will be a complete loss of sensation from the knees downward, and when roots below L 4 are affected, the sensory loss will be limited to the outer and posterior aspects of the legs, to the feet, to the posterior surfaces of the thighs, and to the buttocks. When the sacral nerves alone are injured, the sensory loss is confined to the outer aspect of the dorsal and plantar surfaces of the feet, to a narrow strip up the back of the legs and the upper part of the outer aspects of the legs just below the knees, to narrow areas on posterior surfaces of the thighs, and to the saddle-shaped area on the buttocks. If S 2 to S 5 are involved, then there is no disturbance of sensation of the feet, but the loss is found over the buttocks (S 3 to S 5), over the perineum (S 3), and, in the male, over the scrotum and penis (S 3). The size and shape of the sacral dermatomes are subject to considerable variations but the general outlines were well delineated many years ago by Déjèrine (1914), and recently by Foerster (1936).

CLINICAL SYNDROMES OBSERVED AFTER FRACTURE DISLOCATIONS OF THE SPINE WITH INJURY TO THE SPINAL CORD

1. *Complete Transverse Lesions.* The recognition that an individual is suffering from a complete transverse lesion can be made soon after the injury if the disurbances described on pages 130 to 135 are found. If roentgenograms of the spine show so marked a dislocation of one vertebra upon another that the spinal canal is completely obliterated at the affected level, the cord lesion must be a complete transverse one. If the roentgenograms do not show this extreme grade of deformity, the diagnosis of a transverse lesion must be made from the clinical signs alone. Babinski and Jarkowski and, later, Foerster have called attention to the frequent sparing of sensation in the anogenital region. If sensation is preserved in the sacral dermatomes (especially S 3 to S 5), the lesion of the spinal cord does not involve its entire substance.

I have already mentioned that the disturbances which characterize a complete lesion may occur as the first effect of a trauma to the spine, but that, within a few hours or a few days, there is a return of motor power and of sensation as evidence that the spinal cord was not entirely crushed. Improvement may occur rapidly until most or all of the signs of a cord lesion have disappeared. These are the cases of spinal concussion already referred to. The signs of a complete transverse lesion may be due to a partial injury of the cord with edema of the surrounding cord tissue or to the first effects of a hematomyelia, and in such cases there may be improvement. Unfortunately, in the majority of instances (in over 80 per cent of my cases), an individual with the signs of a complete transverse lesion of the spinal cord found on examination soon after the injury, turns out later to have a complete transverse lesion. This is, however, not a certainty until at least a few days or at the most several weeks have elapsed. In an experience extending over many years I have never seen the signs of a complete lesion after a trauma persist for more than three weeks unless the lesion was a complete one.

The individual who has sustained a transverse lesion is usually severely shocked and does not complain of any pain or discomfort excepting the loss of power and sensation. Bed sores may occur very rapidly (within two or three days) in parts of the body exposed to pressure.

Physiologists have known for many years that after complete transection of the spinal cord in animals, the tendon reflexes return after the primary effects of the injury have subsided. But it was believed that after a transverse lesion in man, the loss of reflexes and flaccidity were permanent. If, as occurred so often in the human being, reflexes reappeared and the limbs below the level of the lesion became spastic, this was taken as evidence that at least some fibers of the cord had been uninjured. The experiences of the World War have given the coup de grâce to the Bastian-Bruns law, and it is now known that in a large proportion of individuals who have sustained a complete transverse lesion above the lumbosacral cord and who survive for a number of months or years, a number of the reflexes return, the limbs become spastic, and automatic emptying of the bladder sets in. However, there is never any return of voluntary motor power or of sensation.

During the first weeks after a complete transverse cord lesion, the only reflex movement that can usually be elicited is a plantar flexion of the large toe upon scratching the sole of the foot; the reappearance of dorsal extension of the toe within the first two weeks is an evidence that the lesion of the cord was not a complete one. Within a few months, however, the lower limbs are no longer flaccid, a gradual increase of stiffness appears together with the reappearance of knee and ankle jerks. It is noteworthy that flexor reflexes usually reappear earlier than extensor reflexes, so that at first the so-called mass reflexes are entirely flexor. These mass reflexes occur mainly in the muscles of the lower limbs and abdomen. In these mass reflexes, which result in the drawing up of the limbs from flexion at the hip and knee, the movement may be so slow that the patient (and in some instances even the physician) may suspect that voluntary motor power has returned. The mass reflexes are often accompanied by profuse sweating and by automatic emptying of the bladder and can be elicited by irritation of the skin of the soles of the feet or of the leg. In this spastic stage, bed sores, if properly cared for, will heal, and the general condition of the patient may be a very good one. The individual may remain in this condition for many months or for years. Sooner or later, however, no matter how carefully the bladder has been cared for, it and later, the kidneys become infected, with resultant irregular fever and loss of flesh. When the infection has advanced to a certain stage, the spasticity of the limbs will become less and the hyperactive tendon reflexes diminished. Finally death occurs from debility and sepsis or from pulmonary complications.

When the transverse lesion is in the cervical cord, the progress of symptoms is often rapid; there is often irregular fever not explicable by bed sores or urinary infection, and rapid emaciation. Death, preceded by medullary disturbances, often occurs within a few weeks. In transverse lesions of the lumbosacral cord or of all of the roots of the cauda equina below the conus, urinary incontinence usually persists. It is often difficult to keep the patient's back dry and bed sores are very apt to occur and to persist. In these individuals (although there are curious exceptions) the lower limbs remain flaccid.

2. *Partial Lesions of the Spinal Cord and Nerve Roots.* The cases may be divided into (a) individuals in whom the early signs are

those of a complete transverse lesion but in whom within a few days motor power and sensation are recovered to a variable degree; (b) those with signs of a lesion of only part of the cord; (c) those with injuries of some of the roots of the cauda equina.

Root pains are much more frequent in the patients in whom only a part of the cord is affected, and these root pains may be so severe that a laminectomy has to be performed for the relief of the pain rather than for the lesion of the spinal cord. After the first effect of the injury has passed, disturbances of the bladder are very apt to disappear unless the lesion involves a considerable part of the transverse diameter of the cord or is lumbosacral or unless there is a considerable degree of hematomyelia (*q.v.*). In these individuals improvement may be rapid so that within a few weeks or months many of the signs of disturbed function can no longer be found, but it is safe to say that very few patients who have had a cord injury recover so completely that some change in reflexes and some sensory disturbance will not remain.

The retrogression of disturbances is often very slow so that several years may pass during which there is slow and steady improvement. The extent of this improvement will, of course, depend upon the nature and extent of the lesion.

In some individuals, the symptoms are so slight that they will be revealed only by careful neurological examination. Walshe and Ross (1936) have called attention to the clinical picture of minor cord lesions in association with injuries of the cervical spine. In these patients the paralysis of the limbs is not very severe and attention is not drawn immediately to the spinal cord injury, because of the fact that the injury was primarily of the skull and produced by violence directed against the head. The common story given by the patients is that when resumption of activity begins after recovery from the immediate effects of the head injury, it is found that the arms alone or both arms and legs are weak. The essential elements in the clinical picture are as follows: "(a) Limitation of movement of the head and neck, with pain and stiffness on movement. (b)

Some general weakness of the upper limbs, sometimes associated with special weakness and wasting of muscles supplied by the fifth and sixth cervical segments. Some hypertonus and slowness of movement may also be found. (c) Characteristic differential changes in the tendon jerks in the arms, usually including inversion of the radial reflex. (d) Slight hypertonus of the extensor group in the legs, with increased knee and ankle jerks, and in some cases ankle clonus and extensor plantar responses. Slight weakness of dorsiflexion and a spastic gait may also be seen. (e) An absence of sensory loss, at least at the time at which the cases have come under our observation. . . . (f) Absence of defects of sphincter control. (g) Slow and imperfect recovery."

As the patients are usually seen several months after the supposed cranial trauma, the condition may be diagnosed as amyotrophic lateral sclerosis, progressive muscular atrophy, or primary lateral sclerosis, owing to the absence of sensory changes and of disturbances of bladder control.

In traumatic lesions of the roots of the cauda equina, the disturbances will depend to a great extent upon the level at which the roots were injured. The lower the injury, the less the loss of motor and sensory function. When only some of the roots have been traumatized, the sensory and motor disturbances may be limited to one limb or to a part of one limb. Thus, only one knee or ankle jerk may be lost, there may be an isolated paralysis of the peronei of one side, or changes in the reflexes may affect parts of both limbs. There may be only slight disturbances of bladder function or micturition may be normal. The disturbances of sensation may be irregular and patchy.

The amount of improvement to be expected after injuries to the roots of the cauda equina depends upon the severity of the lesion of the affected root or roots.

After the expiration of months or years, individuals who have sustained a trauma of the spine with partial injury of the spinal cord, may begin to show progressive symptoms. The symptoms may be due to secondary bone changes with compression of the cord or of a root, to adhesive processes in the membranes, or to a slowly progressive gliosis. The changes in the bony spine (spondylitis or arthritis deformans, *q.v.*) may consist of new bone formation or of the secondary bone atrophy known as Kümmel-Verneuil disease. The projection into the vertebral canal of the partly dislocated body of a vertebra may produce secondary alterations in the cord at the affected level, due to repeated slight traumatism to the cord in movements of the back. Herniation of the nucleus pulposus of an intervertebral disc with the symptoms of this condition may be the result of a trauma. Small areas of hemorrhage or softening in the cord may be the starting point of a spinal gliosis. In these patients it is important to determine whether the disturbances are due to compression of the cord or to intrinsic disease. Carefully done spinal manometric tests will often give valuable information.

Prognosis. The outlook for a patient with a transverse or a marked lesion of the spinal cord due to trauma is always grave, although many individuals with partial lesions recover satisfactorily and others recover sufficiently to be able to resume their earning capacity. The prognosis is graver, of course, in complete than in partial lesions, and graver in cervical than in thoracicolumbar lesions. The results in a series of 314 cases of fractures collected by Frazier (1918),

Table IV

TABLE OF 314 CASES OF FRACTURE AND FRACTURE DISLOCATION ACCORDING TO INJURY OF CORD AND ROOTS (Frazier)

	Complete Transverse Lesion	*Partial Lesion*	*Root Involvement*	*No Involvement of Root or Cord*	*Not Stated*	*Total*	*Percentage*
Recovery..........	0	18	6	33	2	59	19
Improved..........	18	46	8	47	2	121	39
Unimproved........	13	4	2	7	0	26	8
Immediate death....	21	2	0	1	2	26	8
Subsequent death...	39	15	0	3	2	59	19
Not stated.........	2	3	2	14	2	23	7
Totals.........	93	88	18	105	10	314	100

are given in Table IV in which two of Frazier's tables are combined.

There are so many factors that must be taken into account that statistical studies often do not give an adequate idea of results, excepting statistics of death rate of injuries within a few days or weeks of the accident.

With special reference to disability and earning capacity, the following statements may be made regarding prognosis:

The patient with a complete transverse lesion of the cord, if he lives for any length of time, is fully incapacitated for work for the remainder of his life.

The patient with a fracture of the lower dorsal vertebrae and a marked lesion of the lumbosacral cord, may live for years, but the diminution of power in the lower extremities, and especially the total or partial loss of control of the vesical and rectal functions, makes him useless, in the majority of instances, as far as earning capacity is concerned.

The individual who sustained a fracture of one of the lumbar vertebrae with complete crushing of the roots of the cauda equina, may live for many years, but his earning capacity is always greatly reduced, both by the diminution in the power of the lower limbs and by the bladder and bowel disturbances.

The patient with a partial injury to any part of the spinal cord may recover so much of the function that has been lost that he is able to resume his full earning capacity. While it is impossible to make any categorical statement on this subject, because so much will depend upon the amount of injury to the cord, one may say that a reduction of earning capacity occurs in at least 60 to 70 per cent of the patients, due to remaining weakness and secondary atrophy of an upper extremity or of both upper and lower limbs, to persistent bladder disturbances, to secondary deformities, and so forth.

The main amount of improvement after a spinal cord injury will occur within the first six months, and little functional improvement will occur after about one year has passed.

TREATMENT OF INJURIES TO THE SPINAL CORD AND NERVE ROOTS WITH OR WITHOUT FRACTURE OR DISLOCATION OF THE VERTEBRAE

Care must be taken in the handling of an individual with a suspected vertebral and spinal cord injury. In order to prevent possible further dislocation of fractured or dislocated bone with added injury to the spinal cord, the injured person should be kept flat on the back and should be moved without causing any bending of the back. As the bone or cord lesion may be in the cervical region, the head should not be bent on the trunk when the patient is lifted in order to be transported. If the injured person is seen at the place where the accident occurred and a stretcher is not available, a practical plan is to carefully push a board underneath the back and to transport the individual on this board. Such a board can usually be found in the immediate neighborhood.

Aside from the treatment of the fractured or dislocated bone, which is discussed in the following pages, the main care of a patient who has sustained a spinal cord injury, includes the prevention of bed sores, the care of the bladder, and the protection of the urinary tract against secondary infection. The prevention of decubitus demands that the patient be placed on an air or water mattress, and that care be taken to prevent undue pressure upon the back, sacrum, hips, heels, and so on, by the use of cotton pads or rubber rings over the regions exposed to pressure. Good nursing care is of primary importance.

Attention to the care of the bladder is very necessary, for the early occurrence of infection of the bladder can be prevented only by observing the most scrupulous asepsis during catheterization. Despite every precaution, cystitis is only too apt to develop and with it the danger of ascending infection of the genito-urinary tract. So great is the danger of infection that many have recommended that the catheter should not be used, but that the bladder should be permitted to become overdistended and to overflow. Opinions differ regarding the advisability of this method, but it has been the experience of many, including myself, that the tendency to secondary infections in a chronically overdistended bladder is very nearly as great as when careful catheterization and bladder irrigations are used. The insertion of a permanent catheter may accomplish more. Prolonged drainage by suprapubic cystotomy or a permanent catheter combined with some type of drainage and irrigation apparatus such as that suggested by Munro, may be necessary.

Nonoperative Treatment of Fractured or Dislocated Vertebrae

Nonoperative treatment will depend to a great extent upon the situation of the bone injury and the degree of dislocation. Rest and an extension apparatus with counter weights is very useful in cases of recent injury, where reduction of dislocated fragments and immobilization may be accomplished by traction aided by properly applied pillows or a molded plaster of paris splint for support or for the gradual reduction of the dislocation. When one body has slipped over another and the injury is *recent*, methods of reduction by strong traction and careful manipulation will often reduce the dislocation. In order to make sufficiently strong traction on the head in dislocations of the cervical vertebrae, some one of the various types of collar is useful.

A much discussed question is whether attempts at reduction should be made if there are no cord disturbances. I believe that in fresh dislocations, this procedure should always be tried. If roentgenograms show that the dislocation has been reduced, the head, neck, and upper part of the chest should be immobilized in a plaster of paris splint. When this is removed, some type of supporting brace should be worn for at least several months.

If a recent dislocation is more marked, and especially if there are only slight cord disturbances, an effort at reduction should always be made; the patient or family should, however, always be informed that there is a risk that the symptoms may be aggravated and that efforts at reduction may not be successful or may be only partially so.

If the signs indicate a complete transverse lesion, there is no advantage in attempts at reduction of the dislocation, and the manipulations may do great harm if, perchance, the lesion is not a transverse one. There is no general agreement regarding open operations for vertebral dislocations; some surgeons are strongly in favor of open operations in all cases and others are just as strongly opposed.

Operative Treatment

Considered from the viewpoint of the injury to the spinal cord, there is still great difference of opinion among neurologists and neurosurgeons regarding both the indications for operative intervention and the time when an operation should be performed. Many neurosurgeons of experience believe that a laminectomy is indicated in most patients as soon as primary shock due to the injury has disappeared; others of equal experience believe that the surgeon should always wait four or five weeks. Some believe that an exploratory operation is justified even if the symptoms are those of a complete transverse lesion; others recommend surgery only in partial lesions. My own experiences have led me to take a conservative position.

If a patient has a complete transverse lesion of the cord, operative intervention can do no good, and if the signs are those of a complete transverse lesion but the patient turns out to have an incomplete lesion, there is no harm in waiting. The claim so often made, that if the patient has a complete crushing of the cord, the operation can do no harm as the condition is hopeless anyway, and if the patient has not a complete lesion, the operation may do good, is fallacious and should be abandoned.

The only patients with a fresh fracture of the spine in whom operative intervention may be indicated, are those who have symptoms of partial loss of function together with the x-ray evidence of marked deformity of the spinal canal with pressure upon the spinal cord by dislocated bone or bone fragments; and the very rare patients who have severe root pains from pressure of dislocated bone upon one or more sensory roots. That the latter condition is very unusual is shown by the fact that in the not inconsiderable number of cases of fracture of the spine that I have seen in the last twenty years, there was only one case of an incomplete cord lesion in which the severity of the root pain was an indication for surgical intervention.

Only occasionally (I believe in not more than 1 to 2 per cent of patients with incomplete cord transections) does the x-ray show so marked a deformity of the spinal canal that immediate operation is imperative.

In the large majority of instances it is advisable to wait, for the patient can be operated upon one or two weeks later if there be an indication for operation, with a better chance of benefit than immediately after the injury has been sustained.

It is my firm belief, therefore, that only exceptionally should the surgeon intervene early after a fracture of the spine, and that a waiting policy is indicated in almost all of the patients.

LATE OPERATIONS

It is never justifiable to perform a laminectomy after the symptoms and signs of a transverse lesion have existed for many months or for years. I know of several cases in which with the hope of doing some good, a laminectomy was performed under these circumstances. Nothing is ever accomplished by such a procedure and the fact that the dangers of spinal operations are very small should not influence the neurologist or the surgeon in favor of exploratory laminectomy in these cases.

Many months or years after a fracture-dislocation of the spine, there may be increasing signs of compression of the spinal cord or nerve roots by bony overgrowth. In these patients manometric tests will always show a spinal subarachnoid block and a decompressive laminectomy with removal of thickened bone is always indicated. As already mentioned, rupture and hypertrophy of a ligamentum subflavum may give rise, in rare cases, to root pains which will disappear after laminectomy and removal of the affected ligament. Finally, if I may summarize my views regarding justification for operation months or years after the injury, the following statements may be made:

1. Operation may be of benefit a few months after the injury in

patients with persistent spinal symptoms in whom the x-ray shows more or less marked angulation or deformity of the vertebral canal. In these patients, a wide decompressive laminectomy may allow the cord to bulge backward and thus an acute angulation of the cord may be relieved. I have, in some instances, seen a great deal of improvement follow such a decompressive operation.

2. Operative intervention is indicated in those patients in whom there has occurred, months or years after the vertebral trauma, so great a production of new-formed bone, visible on the x-ray films, that there are progressive signs of interference with the functions of the spinal cord due to the increasing pressure of the new-formed bones. In my experience, there have been several patients who were greatly improved by an operation at which the new-formed bone was removed and a wide decompressive laminectomy performed.

3. In a few patients there is persistent pain due to adhesions between the membranes and scar tissue around sensory roots, even though the x-ray examination fails to show any bony abnormality. Some of these patients have so much pain that an operation becomes imperative, if they are not to become drug habitués. In some of them pain may be relieved and spinal symptoms improved after the division of adhesions and constricting bands. But one can never promise the patient certain relief from the decompressive laminectomy and the division of adhesions.

Occasionally, when the root pain is strictly limited to one or two sensory roots, a laminectomy and division of the affected root may give complete relief.

Where pain is the most disturbing symptom and is persistent and very severe, complete relief may follow a laminectomy and division of the anterolateral tracts of the spinal cord (Chapter XVI).

4. Occasionally, after a fracture of the spine with injury to the spinal cord, persistent spasticity of the lower limbs prevents locomotion, although there is good motor power. Such a patient can be put upon his feet again by the division of the proper posterior roots. This operation, based upon the investigations of Foerster, is now rarely performed, but it is a very useful operation in some of the

patients with persistent spasticity of the lower limbs after a spinal injury.

5. In rare instances, in patients with traumatic hematomyelia, in whom marked symptoms persist, or in whom, many months after the injury, an advance of symptoms occurs which is probably due to an increase of fluid in a cord cavity, a laminectomy with incision of the cord and evacuation of the fluid will be followed by a marked improvement in the power of the lower limbs. I have twice seen great benefit from such an operation, although in both instances the bladder incontinence was not improved.

The description of the various steps of the operation of laminectomy is found in Chapter xv. A few remarks may be made concerning some special features of the surgery of the spine and spinal cord in spinal injuries. In fresh spinal injuries care must be taken in the transfer of the patient from his bed to a stretcher and from the stretcher to the operating table. If possible, it is advisable to move the patient in his bed to the operating room. Especial caution must be exercised in turning the patient into the prone position on the operating table, and the surgeon should always be present to instruct orderlies or nurses how to lift and turn the patient. This is a responsibility which should not be delegated to the nursing personnel.

Fragments of bone should always be removed with great care. They should be freed gently from surrounding tissues, the surgeon being especially careful not to make pressure upon the already traumatized cord within the dural sac. If fragments of bone have penetrated the dura, it is advisable to incise the dural sac freely, so that the fragments can be removed with the cord in full view. The same statement holds for the removal of foreign bodies, and the region from which a foreign body has been removed should be gently swabbed with weak tincture of iodine so as to forestall a possible secondary infection.

If a fragment of bone or a foreign body is partially imbedded in the cord, great care is called for. Only by slowness and gentleness of manipulation can the foreign body be removed without causing added injury to the delicate cord tissue. An already traumatized spinal cord is very sensitive to the slightest degree of additional trauma, and it is a safe rule not to touch the cord with fingers or instruments unless it is absolutely necessary. Clots of blood or shreds of tissue should not be manually extracted, but should be washed out of the wound by irrigation with saline solution.

Whenever possible—and this is usually the case—the wound should be closed by careful suture of dura, soft tissues, and skin. The same care should be taken in the transfer of the patient from the operating table to the bed, as was exercised in the transfer from bed to operating table, and the patient should be at once placed upon an air or water mattress. If the spinal fracture was in the cervical region, some variety of splint or collar should be used to immobilize the head and neck.

The foregoing remarks have referred to the comparatively rare instances in which operative intervention is undertaken in fresh fractures of the spine, but it is a safe principle to apply the same rules to operations performed a few or many weeks after the injury.

BULLET AND STAB WOUNDS OF THE SPINAL CORD

Bullet Wounds

While bullet wounds of the spinal cord are met with in civil life, most of our knowledge of these lesions is derived from experiences in war, especially from the experiences in the World War.

Wounds of the spine and spinal cord due to bullets are among the most serious injuries, and injuries due to high explosives are no less so. Wounds of the spinal cord are most frequent in the thoracic region because it is the longest part of the spinal cord, but they occur at every level. In 300 cases of spinal injuries in the World War, reported in 1915 by Gordon-Holmes, the lesion was situated between the sixth and ninth thoracic segments in 65 cases, and between the fourth and the sixth cervical segments in 51 cases.

The injuries to the vertebrae resulting from bullets are varied. They may be limited to one vertebra or may involve a number of the bones of the spine, depending upon the direction taken by the bullet and its speed and power of penetration. The injuries produced by high explosives often affect a large number of the vertebrae.

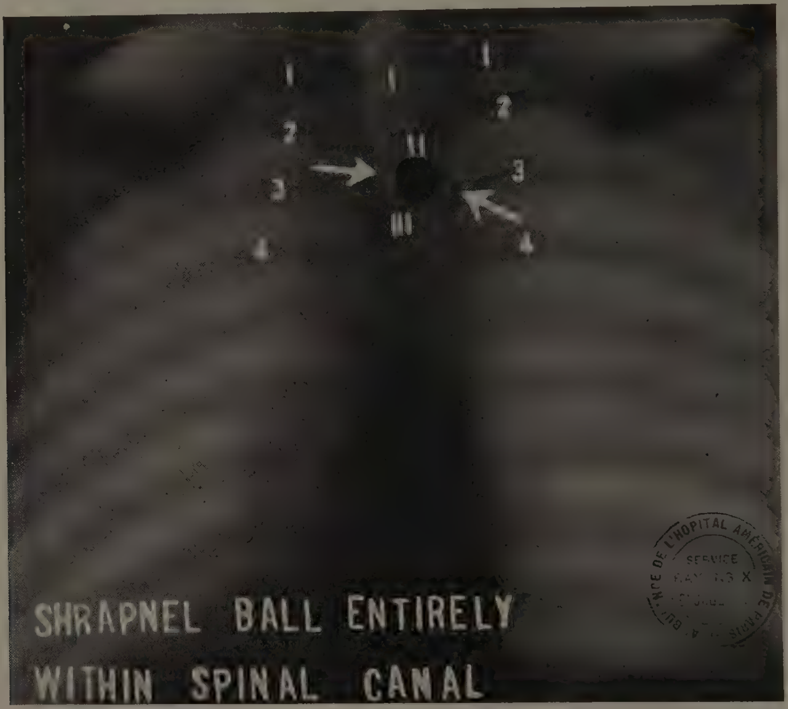


Fig. 85. Shrapnel ball entirely within the spinal canal between the second and third thoracic vertebrae.

With the high speed of the bullet from modern firearms, there may be only a canal through the affected bone, the projectile passing through the spine with injury of the thoracic, abdominal or pelvic viscera, or in the reverse direction. More often, however, there is more or less splintering of bone, which may involve, of course, parts of one or more vertebrae. The bullet may remain lodged in a part of the vertebra or may be found within the vertebral canal outside or inside of the dural sac (Figs. 85, 86).

The injury to the spinal cord may be at the same level as the bone injury or, due to the oblique course of the bullet, at some distance from the point of entry on the surface of the body. Therefore the diagnosis of the level of the injury must be made, not alone from the roentgen evidence of bone injury or of the location of the bullet, but from the signs and symptoms referable to the spinal cord and nerve roots.

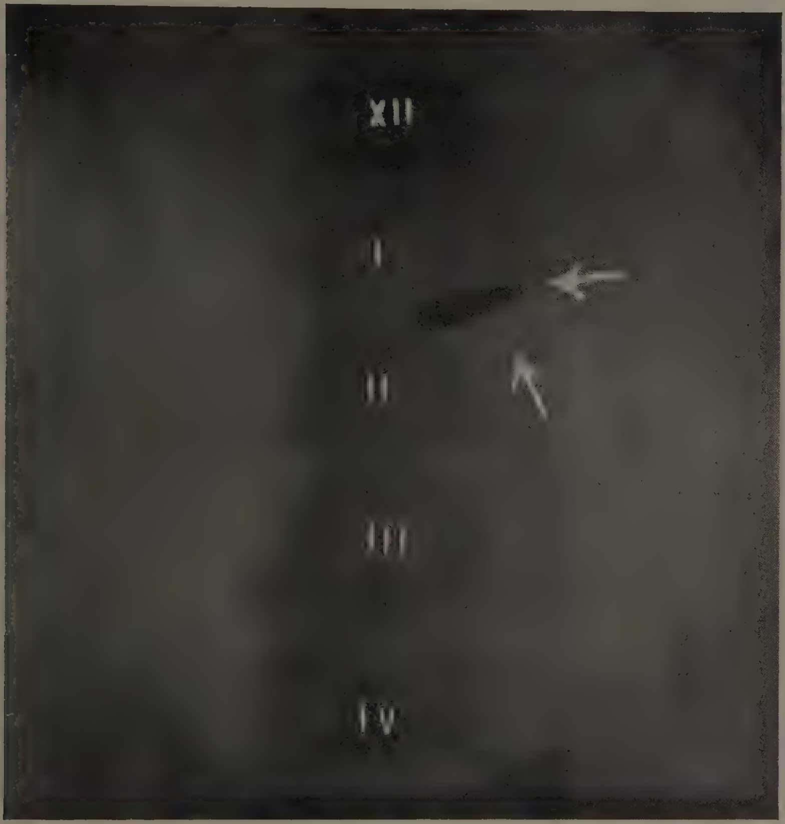


FIG. 86. Bullet which has passed through the spinal canal and is lodged in the first lumbar vertebrae.

In a separate class are the cases in which there are symptoms of a grave lesion of the spinal cord but in which the bullet either did not cause a bone injury or caused, perhaps, only a fracture of a transverse process without other bone injury. These cases may be grouped together with those due to high explosives in which there is no evidence of a direct or indirect injury to the vertebral column, and, in fact, no wound in any part of the body.

When a bullet impinges upon a vertebra without entering the canal or without causing a bony fracture with its effects, the severe concussion may be transmitted to the cord and may cause symptoms varying from slight and temporary disturbances of spinal cord function to a profound disturbance with the signs of a complete transverse lesion. The same result may ensue when an individual has been exposed to the tremendous change in pressure incident upon the bursting near him of a high explosive shell. Some of these individuals have slight spinal cord disturbances which are rapidly recovered from; or the signs of a complete lesion of the cord are found at first, but these are rapidly and entirely recovered from, so that all disturbances have disappeared within a period of from twenty-four hours to one to two weeks. In the individuals who recover very quickly (within twenty-four to forty-eight hours) the lesion is probably edema. If one to two weeks are required for complete recovery, in all probability there were some small, perhaps capillary hemorrhages. There has been much difference of opinion as to the pathological changes responsible for these symptoms of concussion (Hassin, 1923; Lhermitte, 1932; Marburg, 1936; and others) and as to the type of case to which the term concussion (commotio spinalis or *Rückenmarkserschütterung* of German writers) should be applied. I believe it is fair to use the term concussion of the spinal cord whenever one is speaking of the effects of sudden shock and changes of pressure which affect some part of the cord. When the term is used with this meaning, one can include:

1. Cases in which there are, at first, the signs of a complete transverse lesion with complete recovery within a few days or weeks.
2. Cases in which there are slight disturbances which may disappear or may persist.
3. Cases in which there are marked disturbances with partial recovery.
4. Cases in which the signs of a complete transverse lesion persist.

The pathological lesions found in individuals with these different grades of symptoms vary from slight edema to a few small cord hemorrhages to complete secondary myelomalacia.

In all gunshot wounds of the spine with injury to the spinal cord, the cord symptoms vary according to the nature, extent, and site of the lesion. In general, the signs and symptoms are not dissimilar from those found in fracture-dislocations of the vertebrae, but if the projectile has injured only the superficial part of the cord, the evidence of a very localized lesion may be clean-cut. Among others, Riddoch and Head have made careful studies of the disturbances in the reflex functions after wounds of war. In complete transverse lesions, according to Riddoch, three clinical stages may be recognized: (1) The stage of muscular flaccidity; (2) the stage of reflex activity; (3) the stage of gradual failure of the reflex functions of the isolated portion of the cord. The symptoms of these stages have been well described by Riddoch (1917).

As has already been mentioned, the neurological disturbances produced by bullet wounds with partial injury of the spinal cord are in the main similar to those due to fracture-dislocations from other causes. Splinters of bone or the bullet itself, if retained in the vertebral canal, may make pressure upon the spinal cord; but in the majority of instances, from the standpoint of symptomatology the added compression is relatively insignificant. The primary injury to the spinal cord is usually the main factor. In rare instances the course of the bullet has been arrested before the dura has been penetrated and before the cord itself has been traumatized; in these patients, the foreign body may make pressure upon the membranes and, through them, upon the cord.

The *prognosis* after bullet wounds of the spine and spinal cord will depend upon the damage done to the cord and to other important structures in the thorax, abdomen, or pelvis that have been injured. Of course, there is the added factor of danger of infection, both from the foreign body and from particles of clothing that may have been carried into the canal, and danger of meningeal infection from cerebrospinal fluid leakage or a large wound of the soft parts. In bullet wounds of the upper cervical cord, there is added the danger from a lesion of the medulla oblongata. However, with the velocity of the bullet from the modern firearm, infection occurs in a remarkably small number of cases in military and civil practice. In military practice, however, the injuries are often due to shrapnel or high explosive shells, with consequent damage to the soft tissues and therefore greater danger of infection.

There is still considerable difference of opinion among surgeons, as to the patients who should be subjected to immediate operation. For example, Marburg and Ranzi (1917) believe it is essential to wait four or five weeks until the condition has become stationary before a laminectomy is performed. Guillain and Barré (1916), from a study of 100 cases, conclude that every spinal wound should be explored as quickly as possible. Piéri (1917) believes that operation is indicated only when the cord lesion is partial and radiography demonstrates that the projectile is within the vertebral canal.

My views are as follows: The treatment of these injuries is often considered a complex problem, but surgical or nonsurgical therapy must be based upon the principles which guide us in the treatment of all wounds. Unless the patient is in so serious a condition from the spinal injury or from damage to other important viscera that surgery is out of the question, wounds of the soft tissues produced by shrapnel and shells should be treated by débridement so as to forestall infection as far as possible.

As regards the injury to the bony spine and the spinal cord, the indications for treatment are not very different from those recommended in fracture-dislocations of the spine with cord and nerve root injury (*q.v.*). If the patient has the signs of a complete transverse lesion, there is no advantage in immediate laminectomy. Even if the subsequent course should demonstrate that the lesion of the cord is not complete or even if the case is one of concussion (*q.v.*), immediate laminectomy is contraindicated. The same statement holds when the signs point to a partial cord injury. The main damage to the cord is accomplished at the time of the injury and, as already mentioned, compression plays an insignificant part in the production of the cord disturbances. It can do no harm, therefore, to delay any direct treatment of the cord injury, especially if there are thoracic or abdominal lesions which require operative intervention. Even if the spinal cord has been only partially injured, there is no advantage to be gained by immediate laminectomy. On the other hand, if the symptoms and signs indicate that the cord lesion is of slight degree but stereoscopic roentgenograms show that fragments of bone or the bullet itself are so situated as to make pressure upon the cord, a laminectomy should be performed and the splintered bone or foreign body removed as soon as the primary symptoms of shock have disappeared. All in all, no absolute rule can be laid down for the indications for or against immediate surgical therapy. Special indications may be found which might lead the physician to deviate from the general conditions outlined.

Months or sometimes years after a bullet wound of the spine, there may be secondary changes in the bone or in the meninges which demand laminectomy. This subject was discussed in the section on the indications for operative interference after fracture-dislocation of the vertebrae with spinal cord injury, to which the reader is referred for further details.

Stab Wounds

Wounds of the spine and spinal cord produced by sharp or pointed instruments are rarer than bullet wounds. In most instances the instrument is a knife with a sharp blade and the injury is usually inflicted from behind.

The wounds are usually in the lower cervical and upper half of the thoracic region [46 per cent cervical and 54 per cent thoracic in Petren's 93 cases (1910)]. In a comparatively small number of instances the arch or a lamina of one vertebra is fractured by the impact of a blade. More often the bone is simply perforated or the knife blade penetrates the vertebral canal between two vertebrae. As the blow is generally from above downward the injury to the spinal cord is usually at a lower level than the point of entry in the skin of the back. It is not rare for the knife blade to be broken off and for a piece to remain in the canal or in the bone. When the wound is in the cervical region there may be an injury of the nerves of the brachial plexus without any penetration of the vertebral canal, and in these cases one of the large blood vessels is often punctured or divided.

When the instrument has penetrated the vertebral canal, there is generally more or less injury to the spinal cord and this may vary from a superficial wound to a complete section of the cord. Sometimes a part of the cord is divided with almost anatomical exactness. I operated upon a patient, four months after the injury, who had a classical Brown-Séquard syndrome due to a stab wound; a piece of a knife blade was found in the right half of the cord in the midthoracic region.

In stab wounds of the spinal cord the motor, sensory, reflex and trophic disturbances vary with the columns involved and the level of the lesion. Immediately following the injury, there are usually the signs of a transverse lesion, but if the cord has not been entirely divided, the symptoms soon subside, leaving behind the disturbances which characterize a partial lesion.

The surgical treatment must be based upon principles similar to those mentioned in the discussion of the treatment of bullet wounds of the spinal cord.

OBSTETRICAL INJURIES TO THE SPINAL CORD

Obstetrical injuries of the spinal cord as a cause for paraplegia have been known since 1870 when a case was described by Parrot, and thereafter isolated cases were reported from time to time. Crothers (1923) was probably the first to give a clear description of the injury, based upon 5 cases of paraplegia which followed breech extraction. Three years later, Ford (1926) reported 6 cases, 5 of which had been seen during the course of two years.

In the first case of Ford, the lesion was limited to the eighth cervical and first thoracic segments, and to the upper or middle thoracic cord in the others. "The exact upper level was difficult to determine because of the impossibility of a satisfactory examination of sensibility in infants," but sensory loss could usually be demonstrated up to the midthoracic dermatomes. There was usually complete paralysis of the lower intercostals, spinal, abdominal, and lower limb muscles. In no case was there any clinical or roentgen ray evidence of bony injury. In all of the infants, delivery had been difficult and in several excessive force had been employed in extraction.

When the injury is in the cervical region, the lesion is usually fatal at once or within the first few days after birth. When the thoracic cord is affected, the infants may live for a number of months or even for years (Beevor, Burr, Kooy, Crothers, Ford, Byers, and others).

In some of the cases which came to autopsy, the lesion was found to be a complete transverse rupture of the cord with hemorrhage into and around the nervous structures; while in others, the meninges were also torn, and the ends of the torn dura were separated for several centimeters. In the case studied by Byers, in which death occurred four months after delivery, there was extensive degeneration in the cord both above and below the level of the injury.

In most of the infants in which death occurred soon after delivery, the little patient was supposed to have suffered from an intracranial meningeal hemorrhage, but in several of these cases in which an autopsy was performed, the main or only lesion was found in the vertebral canal. If the infant lived long enough, weakness or complete loss of power in the lower limbs and perhaps also in the upper limbs was observed. The tendon reflexes in the lower limbs were usually hyperactive with double ankle clonus. The bowels were usually constipated and there was either retention or dribbling of urine.

In most of the cases reported in medical literature, the lesion was so severe that there was a complete physiological transection of the spinal cord. Lesser degrees of spinal cord injury must occur. Unless the symptoms are marked, they may be missed altogether. It is not beyond the range of possibility that some cases of hemiplegia supposedly due to an intracranial lesion are really instances of a spinal cord injury. Certainly, weakness of an upper extremity, which is not so rarely observed after a difficult occiput or breech delivery, may be caused not by an injury to the brachial plexus alone but by a lesion of both the nerves of the plexus and the spinal cord, and it is not difficult to conceive that the only lesion may be one of the cord. It must not be forgotten that lesser degrees of spinal cord injury which cannot be detected in infants, may give rise to symptoms as the child grows older. The injury to the spinal cord is always due to excessive stretching and not to crushing.

LESIONS OF THE SPINAL CORD DUE TO ELECTRICAL INJURIES

Although, in the majority of instances, the neural lesions produced by lightning are immediately fatal, because of the effect upon the heart and respiratory centers, individuals may recover. Furthermore, with the increased uses of electricity in industry, fatal accidents due to contact with the electric current are becoming more frequent. As far as the spinal cord is concerned, electric shocks may cause lesions which are mainly characterized by hemorrhage with its characteristic symptoms, or by the signs of a complete transverse cord lesion. Foerster refers to a case in which there were both spinal and meningeal symptoms, and similar cases have been described by others. The symptoms may be found immediately after the injury or may develop a number of days or weeks later, and, according to Panse, most often in the cord between the fourth cervical and first thoracic segments. It may be that the strong muscular contractions which are due to the electric current have a relation to the accompanying cord lesions.

CHAPTER V

CONGENITAL ABNORMALITIES OF THE SPINE AND SPINAL CORD

KLIPPEL-FEIL DISEASE

OF RECENT YEARS A GREAT DEAL HAS BEEN WRITTEN CONCERNING THE SO-CALLED KLIPPEL-FEIL SYNDROME WHOSE ESSENTIAL features are a cleft in the occipital bone and cervical arches with maldevelopment and reduction in size of the vertebral bodies. There is often synostosis involving the bodies of the second to sixth cervical vertebrae which are fused into a bony mass (Figs. 16, 17). As a result, the neck appears shortened and many of the neck movements are limited.

The changes in the bodies of the cervical vertebrae, or associated abnormalities of the cervical spinal cord may produce disturbances in the power and sensation of the upper limbs. The motor disturbances are mainly clumsiness or imperfect control of the forearms and hands. Cutaneous and, especially, deep sensibility of the upper limbs may be altered. The diagnosis of the Klippel-Feil syndrome is made from the characteristic changes seen on roentgen films. The syndrome has little practical surgical interest.

Of the congenital abnormalities and defects of the vertebral column and spinal cord, spina bifida is of especial clinical and surgical importance.

SPINA BIFIDA

Spina bifida is a congenital malformation of the vertebral column usually located in the midline, posteriorly. Very rarely, the congenital defect is in the body of one or more vertebrae, anteriorly.

In most instances of posterior spina bifida, the arches of the affected vertebrae are not developed or are absent, or the arrest of development involves also membranes or cord or nerve roots. Through the bony defect there is a protrusion of membranes, and sometimes also of nerve roots or spinal cord.

The incidence of spina bifida is usually placed at about one in each 1000 births.

Spina bifida occurs with about equal frequency in males and females, although in some statistics, the defect was found most often in females. It is most common in the regions where normally union of the edges of the neural groove is latest; namely, in the cervical and, more especially, in the lumbar regions of the spine. In a study of the Surgeon General's Index up to 1905, of 385 operative cases, Moore found that 23 per cent were sacral, 34 per cent lumbar, 24 per cent lumbosacral, 9.5 per cent cervical, and 4.5 per cent thoracic. Two cases were occipital. The relative frequency of lumbar and sacral defects is, however, not more than 70 per cent in most statistics and in a personal series of cases that I have analyzed.

In a small proportion of patients, the lesion is occipital or occipito-cervical; more rarely, there are defects at several different levels of the spine. An extensive defect involving a large part of the spine (rachischisis) occurs very rarely.

Associated developmental defects of other parts of the central nervous system (cerebellum, brain stem and cervical cord) are not unusual.

There has been much confusion as to the nomenclature for the various anatomical types of spina bifida. I believe that the use of terms such as myelocystocele and syringomyelocele should be avoided, and that all cases of spina bifida can be classified as (1) meningocele; (2) myelomeningocele or meningomyelocele; (3) myelocele; or (4) spina bifida occulta. In the first group there is a protrusion of the spinal membranes (dura, or dura and arachnoid, or arachnoid) through a defect in the vertebral arches. In the second group are the cases in which the protrusion consists of both membranes and nerve tissue. In the third group the spinal cord itself is not fully developed and forms part of the protrusion through the vertebral defect.

1. The simplest form of abnormality, although unfortunately relatively uncommon, is the spinal *meningocele* in which the arrest of development mainly involves the bone, with a protrusion of the membranes which form a sac filled with fluid. The wall of the sac may be formed by dura and arachnoid, or the dura extends to the neck of the sac and through a dural defect there is a protrusion of the arachnoid. In the majority of meningoceles, the sac is covered by normal skin, which, at the summit of the tumor, may be thin and translucent.

The sac may be composed of fused dura and arachnoid or these two membranes are not fused and form a single distended sac. If the sac is incised, the normal cord or caudal roots are visible in the canal. There may be fluid between the dura and the arachnoid so that the protruding mass consists of an outer dural and an inner arachnoid sac.

2. *Meningomyelocele or myelomeningocele.* In this variety, the neural tube has closed completely or almost completely, but has remained in close contact with the surface ectoderm. The cord or caudal roots are adherent to the surface of the sac, and the nerve roots may run over the inner surface of the sac or may end in the wall of the sac. Sometimes the central canal of the cord is distended by fluid so that a very thin layer of nerve tissue forms part of the wall of the sac. This form of spina bifida clinically resembles a pure meningocele and is not infrequently encountered.

If the posterior wall of the neural tube, as well as the corresponding part of the meninges, has failed to close completely, fluid may collect and form a cystic tumor which protrudes through the vertebral defect and is covered by normal, thickened or thinned skin.

3. *Myelocele.* In this variety, the neural ectoderm is exposed on the surface of the body because parts of the medullary folds have remained open. The meninges are attached to the edges of the neural ectoderm which forms a zone of parchment skin extending outward into normal skin.

There is usually fluid between the layers of the meninges, and the neural ectoderm is stretched over the surface of the sac. The pressure within the meningeal sac may be so great that part of the normal spinal cord from above is drawn down into the sac so that a normal cord may appear to end in the middle of the sac wall.

In the most marked cases of myelocele a large number of neural segments are involved, there is not any real sac, and the neural tube lies flattened out on the surface in a groove formed by the undeveloped vertebral laminae. The neural tissue has the aspect of a reddish strip which has a moist granular appearance with perhaps a depression in its center. The tissue consists of blood vessels and nerve elements. From this area the nerve roots may be seen to arise and, laterally, the pia and dura mater which become continuous with the skin. In advanced cases, in which there is no sac or cutaneous covering, drops of cerebrospinal fluid may be seen to ooze from the middle of the reddish area.

Von Recklinghausen called the central granular area, the zona medullovasculosa; the area of pia, the zona epithelioserosa; and the adjoining skin area, the zona dermatica.

The cutaneous covering of the sac of the different varieties of spina bifida may consist of normal appearing skin, or skin that is either thickened or thinned. It is not unusual for the skin to be thin and translucent, and ulceration of the altered skin is frequent. The sac may rupture with the escape of fluid, or there may be more or less collapse of the sac and shrinkage of its wall. There may be a port-wine discoloration of the skin or an abnormal growth of hair on the surface of the sac, but this is much more frequent in spina bifida occulta.

It is difficult to gain an exact idea of the relative frequency of the different types. In 170 cases collected by Keiller, 23.4 per cent were meningoceles, and 70.6 per cent were myelomeningoceles, while in the 79 cases of Kolodny, there were 44 meningoceles (56 per cent), and in 57 operative cases of Gross and Sachs there were 20 meningoceles (35 per cent). However, the figures given by Kolodny and by Gross and Sachs were based upon series of patients that were operated upon. If the cases in which surgical intervention was considered contraindicated (in most of whom there were marked paralyses of the lower limbs) had been included, the relative frequency of the meningocele would have been much less. In my own experience, less than 20 per cent of the cases are pure meningoceles. From the point of view of operability and functional results of surgery, this is a matter of prime importance.

Associated Anomalies and Secondary Lesions

Aside from the changes at the site of the spina bifida, there are primary and secondary lesions that may be associated with the spina bifida; namely, harelip and cleft palate, ectopia of the bladder, syndactylism, nonparalytic clubfoot, congenital dislocation of the hip, and so forth. If the developmental anomalies of the central nervous system at and near the site of the spina bifida are excluded, the secondary lesions that are of surgical interest are hydrocephalus, and the so-called Arnold-Chiari malformation of the cerebellum and medulla oblongata.

Enlargement of the head due to hydrocephalus occurs most often with the severe grades of spina bifida—the meningomyeloceles and the pure myeloceles—but it is sometimes associated with uncomplicated meningoceles (Fig. 87). The infant's head may be enlarged at birth or it may rapidly increase in size after birth. The head may appear to be of normal size until a few weeks or months after a plastic operation on the spina bifida has been performed. On the other hand, a moderate degree of dilatation of the ventricles is found so often as a complication of spina bifida, that it is uncertain whether and how often internal hydrocephalus can be produced by the surgical procedure.

Penfield and Cone (1932) have given some evidence that the soft tissues of the sac wall of a spina bifida function as an absorbing structure and that the excision of the sac may disturb the resorption of fluid and thus produce or exaggerate an existing hydrocephalus. The mechanism of internal hydrocephalus in spina bifida has been studied by Russell and Donald (1935). They have drawn attention to the

Arnold-Chiari malformation as a possible cause and they found the malformation in 10 consecutive examples of lumbosacral or dorsilumbosacral meningomyelocele. In 8 of the 10 cases the ventricles were dilated, and in the other 2 cases there was a fistula at the spinal defect.

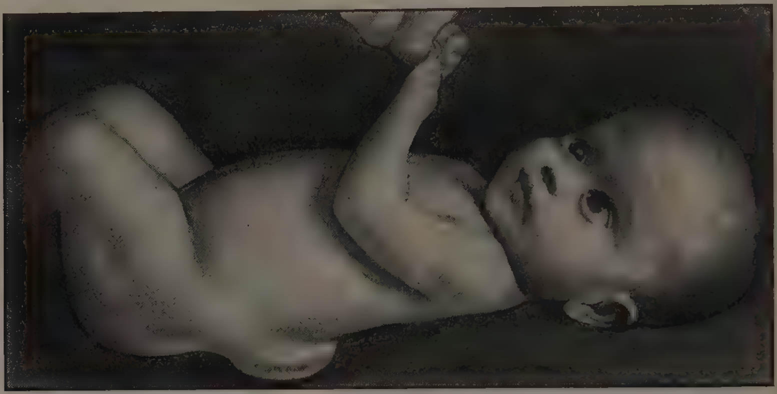
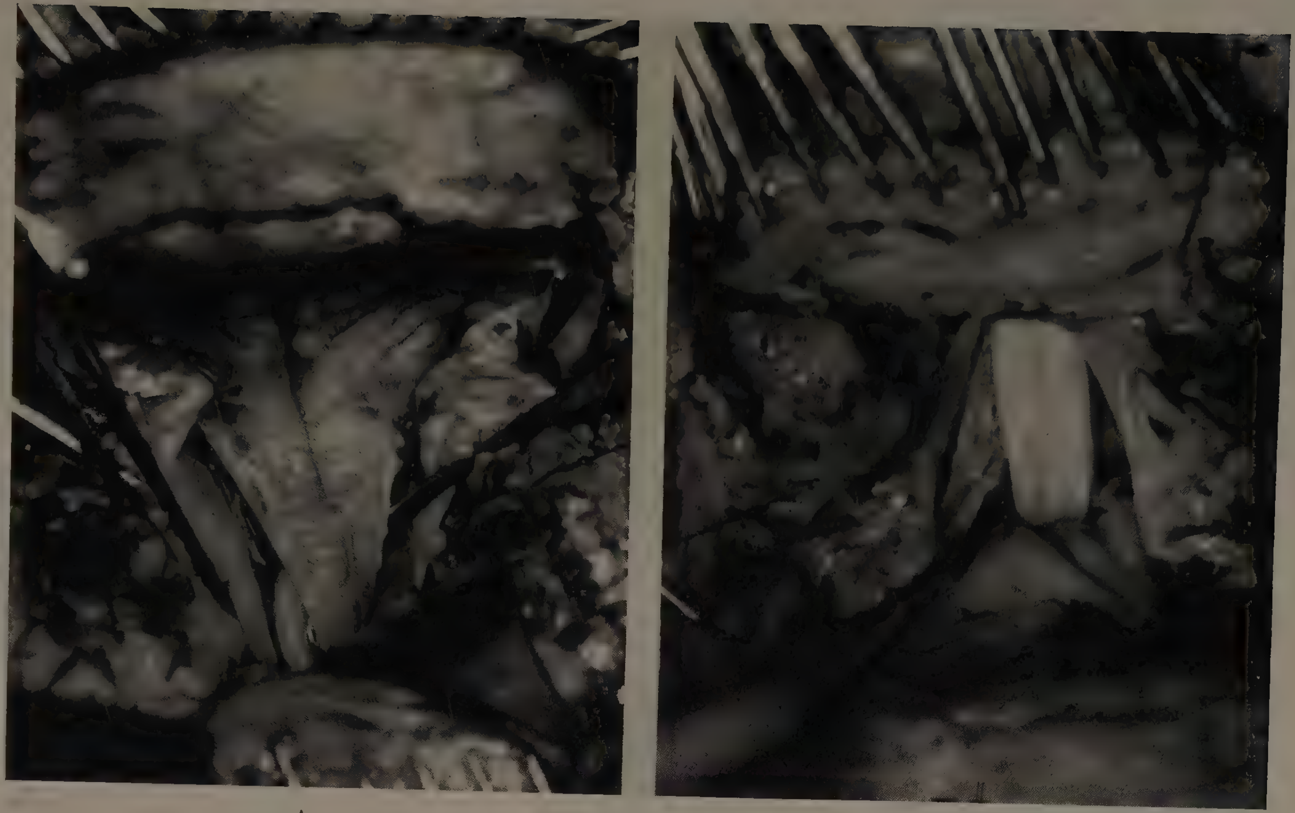


FIG. 87. Spina bifida associated with hydrocephalus. (Shallow. *Annals of Surgery,* page 811, 1928.)

The Arnold-Chiari malformation was first noticed by Arnold (1894); in the following year, Chiari independently described 7 cases. Other cases have been described by Solovtzoff (1901), Schwalbe and Gredig (1907), van Houiveninge Graftdijk (1932), Russell and Donald (1935), Penfield and Coburn (1938). The Arnold-Chiari malformation (Figs. 88, 89) is an elongation into the cervical portion of the spinal canal of the cerebellum and brain stem, which is found with spina bifida. The medulla oblongata is much elongated and protrudes downward into the spinal canal so that a considerable part of the fourth ventricle is below the level of the foramen magnum. The tongue of cerebellum may be adherent to the spinal cord. There is usually hypoplasia of the cerebellum. The cranial nerve roots which arise from the pons and medulla are usually greatly elongated, and many of the cervical spinal roots run in a cephalic direction to reach their exits through the dura mater. As a result of the elongation of the medulla and cerebellum, these struc-



A B

FIG. 88. The Arnold-Chiari malformation. Photographs of suboccipital craniotomy. A. An instrument has been placed under the elongated tail of the cerebellum, which is still attached by its tip to the leptomeninges of the cervical portion of the cord. B. The cerebellar tail has been turned back and the brain stem exposed. The cranial nerves can be seen on the right passing upward into the posterior fossa. (Penfield and Coburn. *Archives of Neurology and Psychiatry*, 40:332, 1938.)

tures are wedged in the foramen magnum and upper cervical canal, and interfere with the free escape of cerebrospinal fluid from the fourth ventricle.

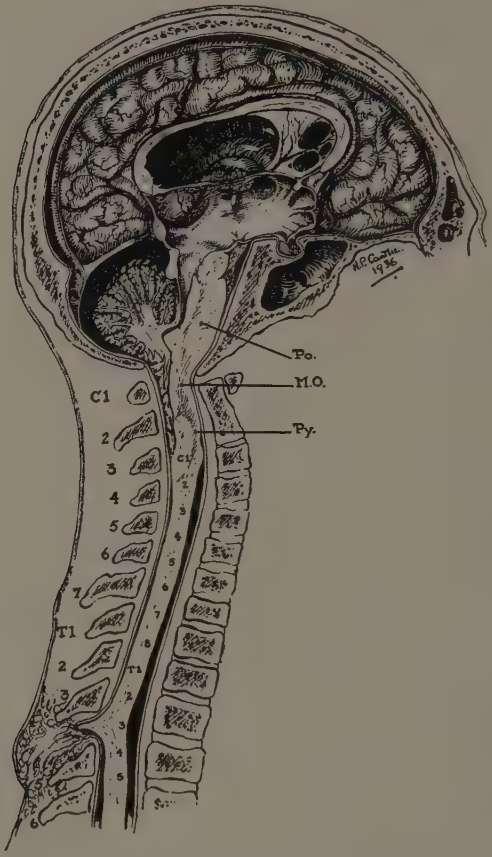


FIG. 89. The Arnold-Chiari malformation. Schematic representation prepared after autopsy. Note the dilatation of the ventricular system produced by closure of the fourth ventricle due to wedging of the cerebellum and brain stem into the cisterna magna. In this Figure *Po.* indicates the pons; *Mo.,* the medulla oblongata; and *Py.,* the decussation of the pyramids. (Penfield and Coburn. *Archives of Neurology and Psychiatry,* 40:333, 1938.)

If pathological investigations of large series of cases of spina bifida demonstrate that the Arnold-Chiari malformation is as frequent as appears from the small series of Russell and Donald, the etiology of the hydrocephalus which complicates spina bifida will have been satisfactorily explained.

SYMPTOMATOLOGY

Aside from the disturbances due to the complicating hydrocephalus, the clinical signs of spina bifida depend upon the situation of the lesion and the degree of involvement of the spinal cord. In the pure meningocele, there may not be any evidence of diminution of power or sensation or alteration of the reflexes, although it is often not easy to be certain of this in very young infants. It is often difficult to demonstrate that the tonus of the rectal sphincter is normal, and the examination may leave one in doubt whether or not there is any area of sensory loss in the lower limbs.

The paralysis is usually flaccid and may involve only one muscle group or a part of a group, or there is total paraplegia. Whenever there are marked motor disturbances, diminution or loss of sensation over sacral or lumbar areas can also be demonstrated. The upper level of these sensory disturbances will depend upon the situation of the spinal defect, but the most marked loss will be found over sacral and lower lumbar dermatomes (buttocks, posterior surfaces of the thighs, outer aspect of legs and feet). There may be paralysis of the vesical and anal sphincters with very little paralysis elsewhere, and in these cases, the absence of the anal reflex and relaxation of the anal sphincter are important signs.

The worst cases are those in which there is complete flaccid paralysis of the lower limbs with marked loss of sensation, and relaxation of the sphincters. When they are first seen soon after birth, these infants may already have more or less enlargement of the head.

It is unnecessary to enter into details regarding the symptomatology of spina bifida. The main signs are the presence of a swelling in the midline of the back, most often in the lumbar or sacral region, with disturbances of motor power of the lower limbs, sensory disturbances (which are difficult to evaluate in infants), and tendon reflex disturbances, especially loss of patellar and Achilles reflexes.

The swelling on the back is usually cystic and in infants (excepting in the closed meningocele), pressure upon the sac will cause increased bulging or tenseness of the anterior fontanelle. Sometimes nerve roots can be felt in the sac, and transillumination may show dark bands running across the wall of the sac. However, one can seldom be certain whether the bands that are felt or seen when the bulging mass is transilluminated are nerve roots or structures made up of connective tissue. If there is a communication between the sac and the spinal subarachnoid space, the sac will be seen to bulge or to become tense when the infant cries or strains.

Even though the spina bifida is cervical, loss of power in the upper limbs is rare. This is probably due to the fact that most of the cervical and thoracic defects are meningoceles. However, it is not unusual for the abdominal muscles to be definitely without tone in many of the cervical and thoracic lesions. The loss of power in the lower limbs varies from dropped foot on one or both sides to complete flaccid paraplegia. The tendon reflexes can usually not be elicited.

In infants, it is always difficult to ascertain the degree of cutaneous sensory loss in the affected limbs, and often one is in doubt whether there is any disturbance at all.

Diagnosis

The diagnosis of spina bifida is always easily made from the local swelling and from the changes in the bony spine found on roentgenograms, excepting in spina bifida anterior in which the protrusion is within the abdomen. As regards anterior spina bifida, it is well to remember that if, in a child or adult with neurological disturbances in the lower limbs or a clubfoot, a cystic tumor is found in the abdomen, the possibility of a spina bifida anterior must be kept in mind.

The more marked the diminution or loss of power in one or both lower limbs, the greater is the probability that one is dealing with a meningomyelocele or myelocele.

In rare instances, sacrococcygeal teratoma or lumbar or sacral lipoma or dermoid may simulate spina bifida, but the differential diagnosis is seldom difficult.

COURSE

An infant with a meningocele and without evidence of any neural lesion, may live to adult age without symptoms. However, few such infants are now permitted to grow up without surgical intervention. If there is a well-marked protrusion, the skin may become ulcerated unless it is well protected against pressure. If the ulcerated skin is very thin, it may rupture with escape of cerebrospinal fluid and infection of the meninges. In the myeloceles without any sac and with a cerebrospinal fistula, meningeal infection is certain to occur, but it is remarkable that infants may withstand a meningeal infection for many weeks. I have operated upon an infant with a ruptured spina bifida in whom the spinal fluid contained many streptococci, but the wound healed by primary union, and the child lived for three months in spite of the fact that the spinal fluid continuously contained the streptococcus. In the majority of cases of spina bifida, secondary complications occur within a few months of birth. Aside from those already mentioned, enlargement of the head due to hydrocephalus is the one most to be feared. Enlargement of the head due to hydrocephalus may be noticeable a few days after birth, or it may begin months or years later. The hydrocephalus may be aggravated after the surgical repair of the spina bifida. When the enlargement of the head has become extreme, a fatal outcome is not long delayed.

OPERATIVE TREATMENT

Indications and Contraindications. There is still considerable difference of opinion regarding the cases in which an operation should be performed and, likewise, little agreement regarding the time when surgery is indicated. Everyone is agreed that the meningocele should be operated upon. It is obvious, also, that the only treatment for a ruptured spina bifida sac is immediate operation.

Some surgeons believe that every patient, even those with complete loss of power in the lower limbs and paralysis of the sphincters of the bladder and rectum, should be operated upon; and these operators quote cases in which, after the surgical intervention, marked paralysis and loss of sphincter control were recovered from. My own experience has been that there is the possibility (but by no means a probability) of considerable recovery of power only when the paralysis of the lower limbs is incomplete. When the loss of power in the lower extremities and loss of control of the bladder was complete, I have never seen sufficient recovery to have made the operation worth while. Therefore I feel that in these infants, unless the parents desire that the effort be made to save the child's life, the surgical repair of the spina bifida should not be recommended. Paralysis of both lower limbs is a great handicap, but when with this there is associated a paralysis of the bowel and bladder, life will not be worth living. When one remembers that such an infant may develop hydrocephalus and may grow up mentally retarded, the arguments against surgery become still more weighty.

The presence of slight enlargement of the head is not a contraindication to operation, but the hydrocephalus must not be extreme. The degree of dilatation of the ventricles is determined by measurements of the size of the head and perhaps by roentgenograms taken after the intraventricular injection of air or oxygen. Not only is the degree of hydrocephalus important but also the rapidity with which the head has been enlarging. If the infant is seen after the expiration of months, and there is the history that the hydrocephalus has existed for several months and that the head has been steadily enlarging, surgical intervention is contraindicated unless the dilatation of the ventricles can be overcome by some other surgical procedure. Attention need hardly be called to the fact that in these infants the mortality of any operation is certain to be high.

I can summarize my views regarding operation for spina bifida by saying that all infants with meningoceles should be operated upon whether or not they have neurological disturbances; that all infants with paralysis of one or both lower limbs should receive surgical treatment unless the paralysis of the limbs is complete. Paralysis of the vesical and rectal sphincters is not a contraindication to operation in spite of the fact that if there is any improvement at all, some lack of control of the functions of bladder or rectum or both will remain.

As regards the time when the operation should be performed, no hard and fast rules can be made. The suggestion that operations be performed only when the infant is 6 to 12 months old is as unjustified as the recommendation that surgical repair should always be made without delay. I have made it a rule to wait until the infant is several months old, unless local conditions (sudden rupture of the sac) made immediate operation imperative. During the waiting period, the sac should be protected against pressure, threatened ulceration of the skin should be treated, and any changes in the size of the infant's head watched for. The condition of the skin in the area of operation may cause delay—sinuses or skin necroses may have to be healed. I have never seen any ill effects from a short period of delay, and there is the obvious advantage that after a few months, the infant will better withstand the operation and that there will be more tissue available for the plastic repair.

Operative Technique. Infants of a few months of age with spina bifida tolerate operation remarkably well if certain precautions are taken and the operation is not too greatly prolonged. Care must be taken that the infant does not lose body heat and fluid. This is accomplished by having the child wrapped in cotton and its body surrounded by warm water bags. Before the operation is begun, it is a good plan to have the infant receive 100 to 250 cc. of saline solution subcutaneously.

For the anesthesia, a minimal amount of ether should be used, and the anesthetic should be started only when the child is in position and the operative area has been prepared. For the operation, the child should be in the prone position with the hips raised and the buttocks covered by a rubber dam fixed to the skin by rubber cement or adhesive plaster (Fig. 90 A). This rubber dam is allowed to remain in place for several days after the operation, and it will help materially in the prevention of soiling of the wound by feces or urine.

The operation should be done as speedily as possible. It is begun

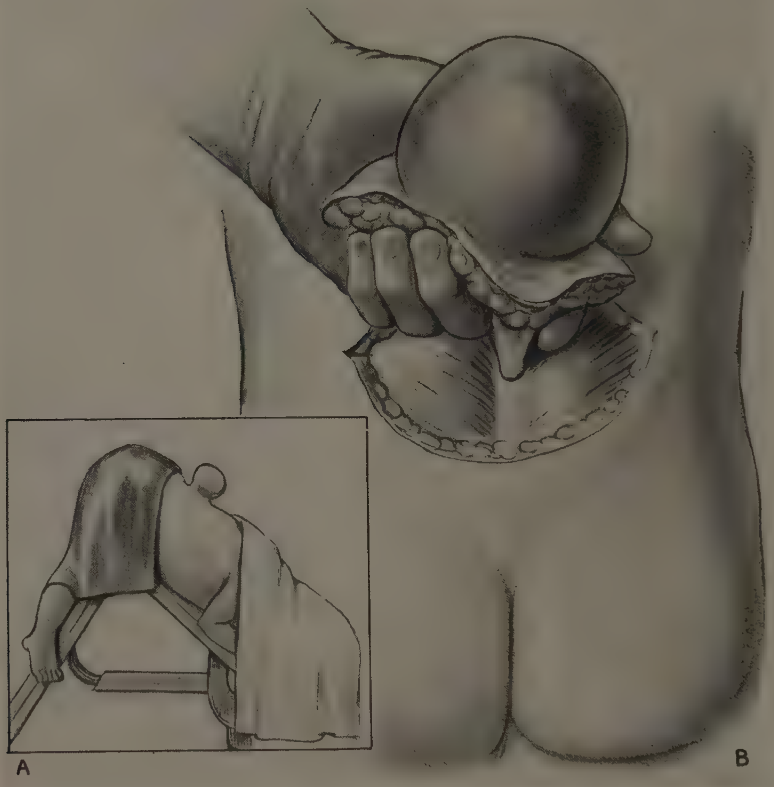


FIG. 90. A. Position during operation, the rubber sheet fastened to the skin. B. Exposure of the pedicle of a spina bifida. (Coughlin. *Annals of Surgery,* page 990, 1931.)

by making an elliptical incision, its long axis transverse, around the base of the sac, or perhaps around the sac near its base (Fig. 90 B). The skin and fat are carefully dissected back until the pedicle is reached over the defect in the spine. Care must be taken not to injure nerves leaving the base of the sac and the surgeon must be on the lookout for large veins which often run near the base of the sac. The sac is then opened, its interior inspected, and the site of cord or nerve roots determined. Nerve elements that can be freed are replaced in the canal. If there are no nerves in the sac wall, as much of the sac as cannot be returned into the canal is cut away and the closure made with interrupted sutures of fine silk. If the condition is one of simple meningocele, the sac may be amputated at its base after the pedicle has been transfixed and tied off by a catgut ligature. In a few instances, I have found the sac of a meningocele entirely shut off from the spinal subarachnoid space, so that it was possible to excise the opened sac without exposure of the subarachnoid space. I have rarely attempted to preserve the entire sac, as suggested by Penfield and Cone (Fig. 91). These authors believe that the lining of the sac is an important factor in the absorption of fluid and therefore that saving the sac may prevent the development of hydrocephalus, but I am not certain that this procedure is essential. Furthermore, in many instances, it is impossible to replace or even partially to replace the entire sac.

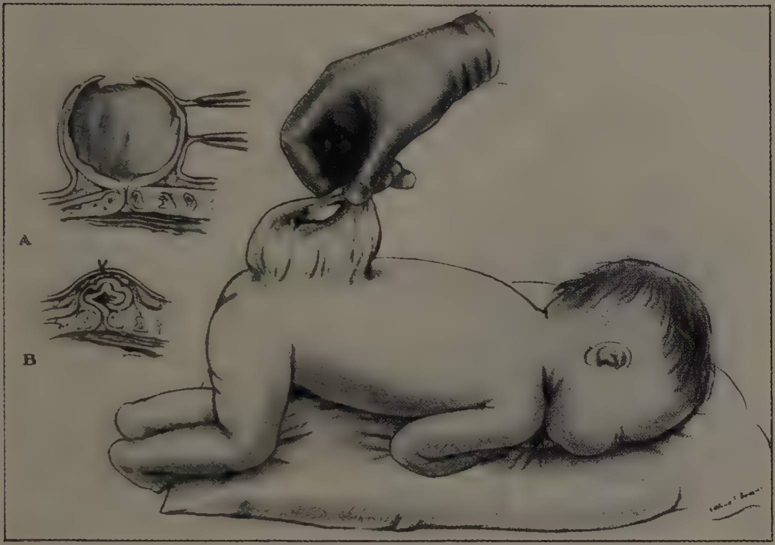


FIG. 91. Sacral meningocele. (Penfield and Cone. *Journal of the American Medical Association,* 98:458, 1932.)

The most difficult part of the operation may be the covering of the defect by plastic flaps of lumbar fascia. Many methods for accomplishing this have been described but the simplest procedure is usually the best. Lateral incisions are made in the lumbar fascia, and flaps of fascia with the base of each toward the midline freed and turned inward to meet in the midline where they are sutured together with fine catgut. It is sometimes possible to have one flap overlap the other. These flaps must be sutured without tension (Fig. 92). When possible, it is advisable to draw the erector spinae muscles together. The closure of the skin is then made transversely. If necessary, a transverse relaxing incision is made several inches above the defect so that the edges of the skin that are to overlie the defect in the bone can be united without tension.

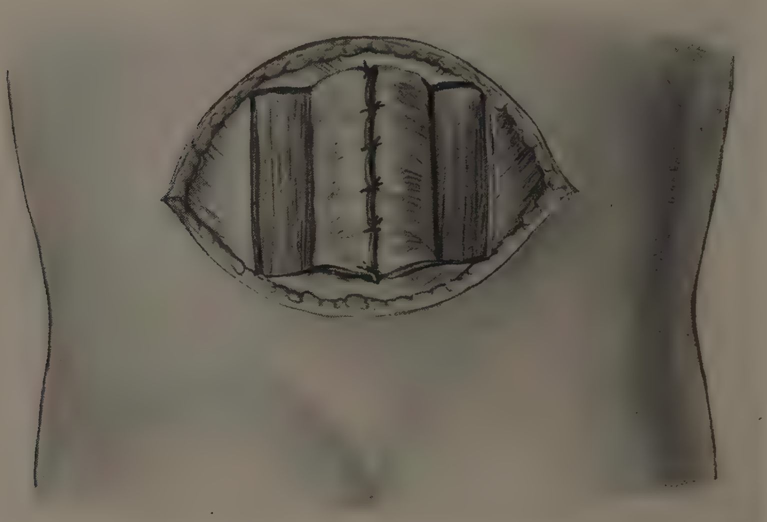


FIG. 92. Two flaps of lumbar fascia of appropriate length and width, the base of each toward the midline and close to the defect, and turned over backward, the one to overlap the other. (Coughlin. *Annals of Surgery,* page 991, 1931.)

After the sac has been incised and inspected, the condition may be found to be hopeless as far as benefit from the operation is concerned. In these patients, the reduction of sac and the plastic operation on the fascia must be completed as rapidly as possible.

The reconstruction of a membranous spinal canal in the presence of a large myelocele with extensive cleft in the vertebral arches is seldom satisfactorily accomplished. Very often, little of the sac wall can be excised on account of the nerve bands running in it. If, in addition, the spinal groove is very shallow, a newly constructed membranous tube will have to remain on the back protected only by fascial flaps and skin united over it.

In the worst cases, there may be so little tissue that it is impossible to reconstruct any kind of a membranous tube or canal. The outlook in such cases is always a hopeless one.

The postoperative treatment is very important. The child should be kept on the abdomen for at least four to five days; this can be accomplished by having the child bandaged to a previously prepared, well-padded plaster of paris mold. Careful nursing is necessary to prevent soiling of the dressings by urine and feces.

Operative Results. The immediate operative mortality was once very high, at least 50 per cent in infants operated upon during the first months of life. In recent years, however, the surgical mortality has been greatly reduced so that at the present time, the immediate mortality is from 5 to 20 per cent (e.g. Gross and Sachs, 1932, 18 per cent; Coughlin, 1932, 8 per cent; Kolodny, 1933, 5 per cent).

The outlook for a satisfactory end result after the surgical repair of a spina bifida will, obviously, depend upon the degree of the congenital maldevelopment of the nervous system. The final results, after surgical repair of the meningocele, are very good: the large majority of my patients recovered entirely or almost entirely, grew up normally, and rarely had any serious disability. In only one case was the head rather large as the child became older.

When nerve tissue is present in the wall of the sac (myelomeningoceles and myeloceles) the results that may be expected are, of course, uncertain. In the meningomyelocele considerable improvement may occur—considerable power in the paralyzed limbs may be regained. As the child grows older, however, some sensory disturbances will be found, there may be a partial dropped foot, and control of the bladder may not be complete. In the worst cases, there will not be any improvement at all.

I am not at all certain how often the operation can be blamed for the occurrence of a complicating and progressive hydrocephalus. It is by no means proved that unless there was already a certain amount of ventricular dilatation, a hydrocephalus will follow a plastic operation on the spina bifida.

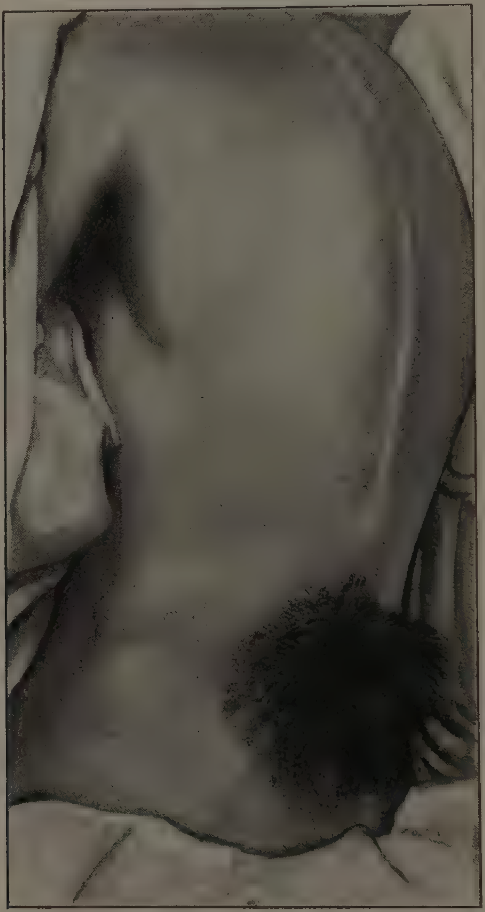


FIG. 93. Hypertrichosis over spina bifida occulta.

SPINA BIFIDA OCCULTA

Spina bifida occulta is a variety of spina bifida, most often met with in young adults, in which there is no true sac and in which there is a fibrous connection between the skin and the spinal cord and its nerve roots through a cleft in the arch of one or more vertebrae. It is well known (Chapter 1) that in embryonic life, the spinal cord extends to the bottom of the vertebral canal; at birth, the tip of the conus lies at the level of the third lumbar vertebra and in adult life at the level of the first lumbar vertebra. In spina bifida occulta, there is a bony defect and fibrous adhesions between the soft tissues of the back and the nerve roots or cord, so that, with the growth of the bony vertebral canal away from the lower end of the spinal cord, there is increasing traction upon nerve roots and cord. The cleft in the bone is generally closed in by a thick fibrous membrane which is continued as a thick band to the under surface of the skin. The skin over the affected region may be normal in appearance, but more often there are marked changes. There may be only a dimpling or slight depression of the skin which may be wrinkled, pigmented, or coarse. There may be a brown or port-wine colored appearance of the skin with the pressure of an increased number of hairs in the affected area. Or there may be a profuse growth of hair (Fig. 93). There is often a lipoma in the affected area (Fig. 94), which may be connected by a fibrous band with another mass of fat within the spinal canal.

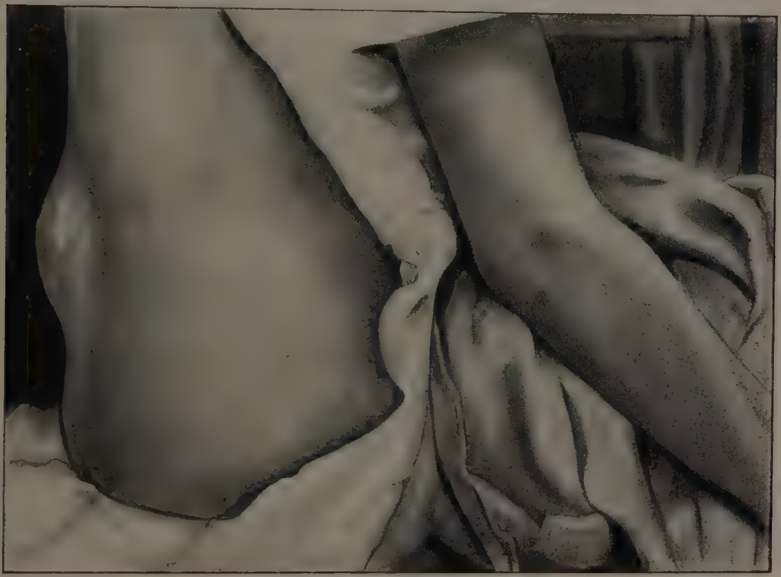


FIG. 94. Congenital lipoma over spina bifida occulta.

SYMPTOMATOLOGY

The symptoms of spina bifida occulta may occur at an early age, but they are usually first observed after the ninth year of life and most frequently in young adults. There may be motor and sensory disturbances in the lower limbs, but the physician is usually consulted because of incomplete control of the function of the bladder (and more rarely of the rectum) or incontinence, or because of trophic changes in the lower extremities (Fig. 95).

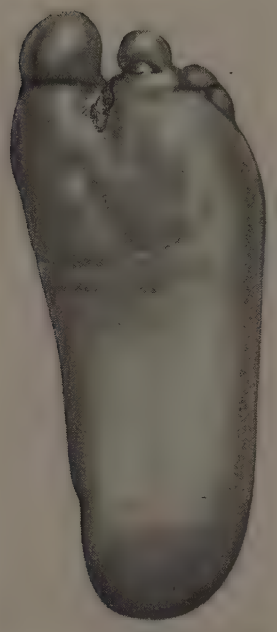


FIG. 95. Plantar ulcers at base of third toe and over the head of the second metatarsal. Note stumps of toes.

Most of the disturbances which occur in spina bifida occulta are due to compression of or traction on the spinal cord and roots; therefore these symptoms are not apt to occur before the tenth to twentieth year, but nocturnal enuresis in children may be due to this cause. In the absence of external signs or pressure symptoms, the condition may pass unrecognized for many years. It is probable that a slight degree of occult spina bifida is more frequent than is ordinarily supposed, perhaps as frequent as bifid lumbar or sacral spines which are so often seen on roentgen films.

The disturbances usually appear gradually, although in a few instances, dropped foot or other motor weakness occurs suddenly. Atrophy of the muscles of the thighs and legs may be the first symptom noticed, perhaps with diminution or loss of one or both patellar or Achilles reflexes. Diminution or loss of sensation over lumbar or sacral root areas may be found but often the sensory disturbances are slight and vague.

The vesical disturbances vary from a slight urgency of urination to complete incontinence. I have operated upon two patients whose only symptom was urgency of urination and partial incontinence.

In the majority of patients I have observed (and most of them were of the female sex), persistent and recurring ulcers on the toes were the only symptom. The trophic sores occurred most often on the plantar surfaces of the toes or the soles of the feet.

The ulcers were difficult to heal and recurrence was usual. The pain from the ulcers was often severe, so that amputation of several toes had been performed before the patients came under my care. In several of my cases, there was a rise of temperature to 104° F. or more before each fresh ulceration occurred.

Clubfoot or other congenital deformity is often associated with spina bifida occulta.

In any young adult, who develops disturbances of urination or defecation for which no other cause can be discovered, roentgenograms of the lower spine should be taken, in order to demonstrate or exclude spina bifida occulta.

The same is true in any child or young adult in whom there are trophic ulcers of the toes or heels. The lower back should always be carefully examined in these individuals. A dimpling of the skin or slight hypertrichosis is of significance. X-ray films of the lumbar and sacral spine should always be taken.

TREATMENT

Only those cases which give rise to symptoms may require surgical treatment, and the surgeon must appreciate that he can never be certain whether, and if so, to what extent, symptoms such as bladder or rectal disturbances can be relieved and whether trophic ulcers will heal.

If operation has been determined upon, a longitudinal incision is made in the affected area and deepened until a lipoma is reached or a tough fibrous band is encountered. This band is freed on all sides and isolated down to the point where it enters the cleft in the vertebral arches. Part of the fibrous tissue in the cleft is carefully excised until normal dura is exposed. In order to accomplish this, the laminae of one or several vertebrae may have to be removed. The fibrous band is then excised at the dura or an incision in the dural sac is made to one side of the attachment of the fibrous band, and the cord and nerve roots examined. If no roots are adherent to the band, that part of the dura that forms the base of the fibrous band is removed and the opening closed by suture. In this way marked traction on cord and nerve roots may be relieved. Sometimes (as in one of my patients) an intradural lipoma was exposed as soon as the dura had been incised. Occasionally, one can find several fibrous bands which are angulating several nerve roots. These should be divided or excised.

The results of operations for spina bifida occulta vary greatly. If it is possible to relieve all traction on and compression of cord and roots, sensory and motor disturbances may disappear entirely and trophic ulcers may heal. In most of the patients I have operated upon, the trophic ulcers healed, but in several of the patients, toes had later to be removed. Cases are known in which amputation of the foot or leg became necessary.

In my experience, long existing bladder disturbances due to spina bifida occulta have been little benefited by surgery.

CHAPTER VI

INFLAMMATORY DISEASES

EXTRADURAL SPINAL ABSCESS

SUPPURATIVE PROCESSES IN THE SPINAL EPIDURAL SPACE OCCUR EITHER AS EARLY OR LATE METASTASES THROUGH THE BLOOD STREAM from an inflammatory process in some other part of the body, or as a direct extension into the spinal extradural space from a contiguous infectious or suppurative process. In some instances, however, no cause for the occurrence of an extradural abscess can be discovered.

The infection through the blood stream may occur in bacteriemia from any cause, and it is frequent as a complication of acute or chronic osteomyelitis of the long bones and of suppuration in the muscles of the back or of some part of the body remote from the vertebral canal. Why extradural spinal abscesses are so often secondary to furunculosis has not been satisfactorily explained. Among more unusual primary diseases may be mentioned actinomycosis and suppurating echinococcus cyst, chronic bronchitis, lung abscess. Extradural abscess may follow penetrating bullet or stab wounds of the vertebral canal.

In the majority of instances, in my experience, extradural abscess occurred as metastasis from inflammatory lesions in some other part of the body, and those who have investigated the reported cases in the literature of the subject have arrived at the same conclusion. Campbell, in 1937, collected 185 cases and found that in 120, the primary lesion was metastatic, while of the remaining 65, the primary lesion was uncertain in 34, and trauma was given as the cause in 26.

The extradural inflammatory process may run an acute or a subacute course. After a preceding inflammatory process in some other part of the body, the symptoms referable to the spinal cord appear suddenly. These usually consist of severe and localized pain in the back with fever, followed in one or a few days by a rapid loss of power in the lower limbs or in all four extremities with more or less marked sensory disturbances and urinary retention or incontinence. The symptoms may begin as weakness of one extremity, but the advance is very rapid, so that in from twenty-four to forty-eight hours there may be flaccid paraplegia with loss of tendon and cutaneous reflexes and of control of the bladder. The spinal symptoms are partly the result of edema of the cord and perhaps the toxic effect of the infection, but mainly they are the result of pressure. Many of the patients are first seen by the neurologist or neurosurgeon when there is already spastic or flaccid paraplegia.

In the majority of instances, pain in the back and marked tenderness during movements of the spine or on pressure over the spinous processes at the level of the lesion are the outstanding disturbances. Cutaneous hyperesthesia may be found even before there are any changes in the reflexes, in power, or in sensation. Due to the pain and tenderness, the affected part of the spine is held rigid. Occasionally, severe root pain is associated with the pain in the back, or it may appear alone in one or both upper or lower extremities. Within a period lasting from a few hours to several days, signs of sensory or motor disturbance of one limb or of several limbs appear. Sensory disturbances may occur before motor, as in one of my patients with an abscess at the level of the fourth thoracic vertebra, who first complained of numbness of the right lower and then of the left lower limb, followed by ascending numbness of the body, before any weakness of the limbs had been observed. On the other hand, I have seen and operated upon several patients with spastic or flaccid paraplegia but without or with only very slight evidence of diminution of sensation over the affected limbs.

In the stage in which most patients are seen there is often marked rigidity of the neck, and, especially if the lesion is in the cervical spine, a Kernig sign.

Roentgen films of the spine usually do not show anything abnormal. As in all infectious processes, the examination of the blood often shows a leucocytosis.

In most of the patients in whom the signs of a cord lesion are advanced, lumbar puncture and manometric tests will show marked or complete subarachnoid block, with increase of cells, and of globulin and of total protein. When the patient is seen very early, however, there may not be any subarachnoid block, the fluid may be yellow, and cells and total protein above normal. The cell count may be normal while the total protein is increased. In my patients the total protein varied between 100 and 500 or 800 mg. per cent and often clotted spontaneously.

Unless there are evidences of infection of the subarachnoid space and of diffuse meningitis, organisms are not found in the spinal fluid.

The organisms most often found in acute epidural abscess are staphylococcus aureus or streptococcus, more rarely the diplococcus of pneumonia or bacillus pyocyaneus.

DIAGNOSIS

The only treatment for acute extradural abscess is laminectomy and drainage, and the results of therapy depend entirely upon early diagnosis and operation. Therefore recognition of the disease is of the greatest importance.

In a patient with bacteriemia and with severe toxic symptoms, delirium and high fever, the occurrence of a metastatic abscess may not be recognized until much loss of power in the limbs has occurred. But in all conscious patients who have suffered from furunculosis or who have recently had an inflammatory process in some part of the body, or who have had a suppurating focus somewhere, especially a chronic osteomyelitis, the occurrence of severe pain in the back should at once make the physician think of a metastatic epidural abscess. Likewise, the signs and symptoms of an osteomyelitis of the vertebrae, or the presence of a sinus of the back discharging pus, should make one be on the lookout for possible infection of the spinal epidural space. In any individual in whom an acute attack of fever with severe pain and tenderness in some part of the back, is soon followed by disturbances of the functions of the spinal cord, the possibility of an extradural spinal abscess in which an emergency laminectomy may be necessary, must be kept in mind.

If the extreme urgency of such symptoms were more generally recognized, and the surgeon was called in earlier, the results of operation would be much better than they are at the present time. It has been my personal experience that much valuable time is often lost in watching the patient and waiting for the appearance of marked changes in tendon reflexes, power, and sensation.

The importance of early operation is very great, and the correct diagnosis can be made early in the majority of instances. Acute myelitis on an infectious basis can usually be excluded without difficulty. It is a rare condition and does not cause the severe pain and tenderness found with acute suppuration in the epidural space; there is usually not a subarachnoid block by manometric tests. It is rare for an acute vertebral osteomyelitis per se without an inflammatory process in the meninges or on the outer surface of the dura to cause any distinct spinal cord symptoms.

Treatment

The operation consists of laminectomy with incision and drainage of the abscess. Pus is often seen as soon as one or several laminae have been removed, but not rarely, an inflammatory mass is seen to cover the dura and careful incision into it will reveal purulent fluid. Care should be taken that the dura is not injured. One or several drainage tubes are inserted, and after careful toilette of the wound, the soft tissues closed around the drainage tube. In the majority of instances the pus is found without difficulty either on the posterior or lateral aspect of the dural sac.

The results of operation in acute extradural abscess are excellent if the patient is operated upon early, before flaccid paraplegia has occurred. Even with flaccid paraplegia, complete or almost complete recovery of power may occur, as I have seen in several instances. However, in most of the patients with flaccid paraplegia before operation, there is not any or only very slight improvement after the drainage of the abscess.

Of 88 patients who were operated upon, collected by Campbell, 31 or 35.2 per cent died, 23 or 26.1 per cent recovered incompletely, 34 or 38.6 per cent recovered completely. Some of the patients succumbed to their primary disease, others to secondary infections in the meninges, and still others to complicating generalized sepsis or to pneumonia. In almost 40 per cent of the patients I have operated upon, the recovery was complete or nearly complete.

INTRADURAL SPINAL ABSCESS

This condition may follow a bullet or stab wound of the spine, but with the high velocity of the modern bullet, it is rare in the former wounds. An abscess within the dural sac inside or outside of the arachnoid may occur as a localization of a diffuse meningitis, or following a wound infection. I have seen one case in which a localized abscess followed a spinal operation done elsewhere, and one in which a cause for the abscess could not be discovered. In both instances, the symptoms and signs were indistinguishable from those which occur in extradural suppuration. The symptoms and signs which pointed to a localized lesion were clear, and the operation in each instance consisted of laminectomy, incision of the dura, and drainage. One of the patients recovered.

PACHYMENINGITIS AND CHRONIC EXTRADURAL INFLAMMATORY PROCESSES

In every epidural inflammatory process there is a certain amount of involvement of the dura and therefore a certain degree of pachymeningitis. A primary inflammatory process of the dura alone is a rare condition, as is the disease described many years ago by Charcot, which he called "hypertrophic cervical pachymeningitis." In primary pachymeningitis there is a slowly advancing inflammatory process which results in localized or more diffuse thickening of the dura. The thickening may be so localized as to form an actual tumor which causes more or less marked compression of the spinal cord. According to Charcot, the lesion is found most often in the lower cervical region but considerable areas of the dura may be involved. The symptoms of hypertrophic pachymeningitis vary with the location and extent of the inflammatory process. Early and persistent root symptoms—radicular pain, radicular hyperesthesia or anesthesia—occur very often. When the cervical dura is involved, the patient complains of pain in the back of the neck and of neuralgic pains in the upper limbs along the course of the ulnar and median nerves. Weakness and atrophy of the muscles of the upper limbs are early symptoms.

The advance of the disease is usually a slow one; many months or years may elapse before the thickening of the dura becomes great enough to cause compression of the spinal cord. The clinical picture is very similar to that due to an extramedullary tumor at the same spinal cord level. The diffuse character of the early root symptoms, often involving the limbs of both sides as well as of a number of spinal roots, is somewhat different from that ordinarily found in cord compression by tumor, and is more like that found in spinal adhesive arachnoiditis.

Because hypertrophic pachymeningitis *sui generis* is a rare condition, the history of one case I have observed may be given:

A woman, 32 years of age, had had at 10 years of age, some spinal trouble, on account of which she was in bed for a number of weeks. Three years before admission to the hospital, she began to suffer from attacks of pain in the upper part of the chest and shoulders. The attacks of pain continued, and five weeks before admission, she had difficulty with her gait and had a girdle sensation around the upper part of her chest.

The patient was a healthy looking female who walked with a markedly spastic gait. Cranial nerves and upper limbs were normal. Abdominal reflexes were not elicited. The lower limbs were very spastic, and the right lower extremity weaker than the left. The tendon reflexes in the lower limbs were hyperactive with double ankle clonus and Babinski. There was marked diminution of tactile, pain, and temperature sensibility below the fifth thoracic dermatome, with thermal loss below the knees. The sixth thoracic spine was sensitive to pressure.

At the laminectomy, the dura was found to be thickened and congested and very firm and hard underneath the fourth to seventh thoracic vertebrae. At the level of T 6 the thickening was most marked (Fig. 96), and the entire thickening formed a tumor mass which was found markedly to compress the spinal cord. The thickening extended laterally to the dural openings. A piece of dura which measured 5 cm. by 3 cm. was excised. The pathological report of the excised mass was "chronic inflammatory tissue."

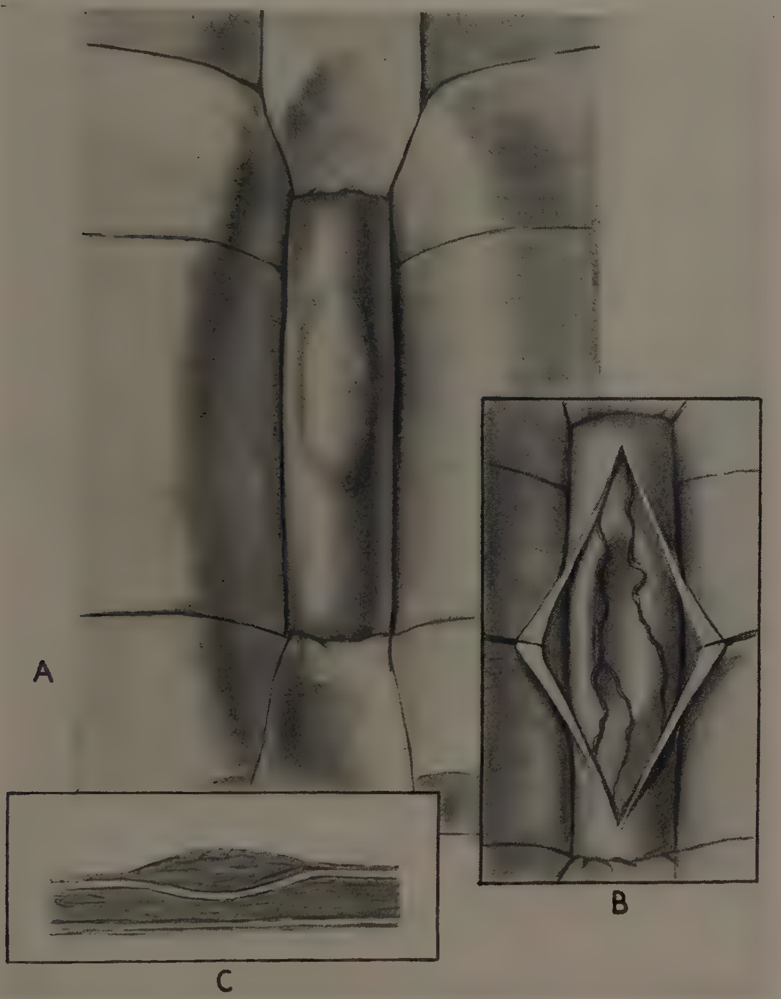


FIG. 96. A. Localized pachymeningitis. B. The thickened dura edges held open and the depression in the cord. C. A diagrammatic sketch of the thickened dura which compressed the cord.

Recovery from the operation was uncomplicated, and the symptoms and signs gradually grew less until she was finally entirely well.

More often the process in the dura is a part of an extradural inflammatory process, which gradually becomes more marked and finally produces the symptoms and signs of compression of the spinal cord. Unless a primary neighboring inflammatory process exists, this usually cannot be distinguished from compression by a true neoplasm.

This chronic inflammatory lesion may follow chronic osteomyelitis of the vertebrae and is frequent in Pott's disease of the spine (Chapter XIII). At any time during the course of the affection, suppuration in the center of the inflammatory mass may occur. If secondary suppuration is due to the ordinary pus-forming organism (staphylococcus aureus) the symptoms may be those of an acute extradural abscess. If the organism is of a low degree of virulence or is some organism such as the tubercle bacillus, the abscess may not cause fulminating spinal cord symptoms, but may cause only an increase in size of the inflammatory mass and increasing signs of compression of the cord often indistinguishable from compression due to a neoplasm. Thus, in several patients upon whom I have operated, in whom the preoperative diagnosis was spinal cord tumor, an inflammatory extradural mass was found which could be peeled from the dura and excised.

When the roentgen films of the spine show that there is tubercular or other bone disease, the cause for the spinal symptoms is naturally sought for as a secondary inflammatory process, but in the absence of x-ray changes in the spine, the diagnosis of the cause of the compression will sometimes remain in doubt.

In these chronic extradural inflammatory lesions, lumbar puncture and manometric tests usually show more or less subarachnoid block with the characteristic changes in the protein content of the spinal fluid; but if pleocytosis is present one should make a renewed survey of history and physical findings before arriving at the conclusion that the symptoms are due to compression of the spinal cord by neoplasm.

When a laminectomy is done for an extradural inflammatory process, the vascularity of the soft tissues and bone will often be found to be unusually marked, so that bleeding from the division of soft and bony tissues is unusually troublesome. Sometimes the inflammatory mass can be peeled off from the dura, and in such a case, the surgeon must be careful not to injure that membrane. In many cases, the dura itself is involved in the process, so that only part of the inflammatory tissue can be removed. It is inadvisable to incise the dura sac, and when the soft tissues are closed by suture, a tube or gauze wick should be inserted for drainage.

SPINAL ARACHNOIDITIS (MENINGITIS SEROSA CIRCUMSCRIPTA SPINALIS, CHRONIC SPINAL MENINGITIS)

This is a condition in which changes in the spinal cord are associated with adhesive processes in the leptomeninges. The disorder is said to be distinctly more common in males than in females, but in my series, men and women were about equally often affected, all of my patients were adults, and the adhesions were found most often in the cervical or thoracic portions of the spinal cord.

It is well known that adhesions between pia, arachnoid and dura occur secondarily in many diseases of the central nervous system—in primary and secondary degenerative diseases of the spinal cord, in syringomyelia, multiple sclerosis, acute anterior poliomyelitis, tabes, and spinal syphilis.

Adhesions of the arachnoid to dura and pia mater occur also with local lesions around the spinal cord, such as tumors, and they may follow trauma to the spine. Marked arthritis of the vertebrae may be followed by adhesive processes in the meninges.

Inflammatory processes in the meninges occur in extradural and intradural spinal abscess and after a primary meningeal infection such as purulent meningitis. Localized adhesions may remain after bullet and stab wound, after operations on the spinal cord, and sometimes after the injection of lipiodol.

The etiology of adhesive processes between the membranes is, therefore, clear in numerous instances; but there are many cases in which there is no preceding history of disease or trauma, and in which, at operation or at postmortem examination, adhesions between the arachnoid and the other membranes are found.

Horsley (1909) described a series of cases which he supposed were gonorrheal in origin, but the evidence he presented for this etiology was unconvincing. According to many authors (Mauss and Krüger, 1918; Rademaker, 1938; and others) trauma is an important etiological factor, and I have obtained a history of injury to the back in one-third of my patients. If the injury was sufficiently severe to cause a vertebral fracture, a connection between the trauma and the spinal lesion is probable, but when the injury was slight without roentgen evidence of a lesion of the bones or ligaments and without disturbances in the functions of the spinal cord or nerve roots soon after the injury, the connection between trauma and arachnoid adhesions is uncertain. A preceding attack of meningitis due to the meningococcus or some other organism or to an infection secondary to disease of the nasal sinuses or of the middle ear may, of course, leave behind an adhesive arachnoiditis.

My analysis of our own cases and of the cases in the literature have led to the conclusion that primary spinal arachnoiditis is very rare. Without doubt, in patients with spinal cord disturbances which lead to laminectomy, adhesions between the membranes without any gross evidence of a spinal cord lesion are often encountered, but the evidence that the meningeal adhesive process is a lesion *sui generis* is rarely conclusive. It is possible (but highly speculative) that repeated traumata during movements of the spine, to a part of the spinal cord fixed by adhesions, may result in marked intramedullary changes.

Pathology

The gross changes in the membranes ordinarily found at operation consist of cloudiness and thickening of the arachnoid with bands of adhesions between arachnoid and pia and arachnoid and dura. These changes most often occur on the posterior aspect of the spinal cord and may fix the cord to the dura. The adhesions are either fine and easily separated, or dense bands divide the subarachnoid space into loculi which contain fluid. The adhesive process may be localized to a small part of the cord or may extend over a large part of the spinal canal. The process may be limited to adhesions between the arachnoid and the nerve roots (Selinsky, 1936).

The spinal cord may have a normal appearance or it may seem shrunken and its color pale. The surface blood vessels may appear smaller than usual, or one or more small veins, compressed by a fibrous band, are enlarged. If fluid is retained within closed cavities formed by adhesions, these so-called leptomeningeal cysts may cause a more or less marked depression in the spinal cord.

SYMPTOMATOLOGY

Leptomeningeal adhesions may produce symptoms by interfering with the free flow of cerebrospinal fluid in the subarachnoid space, or cystic collections of fluid may cause the symptoms and signs of cord compression.

In many instances, the spinal cord disturbances are due to the coexisting (or possibly the primary) intramedullary lesion. As the adhesive process may affect a localized part of the spinal cord or may be diffuse, and as the lesions may be found at any level of the spinal cord or cauda equina, the clinical picture is very variable.

Although the onset may be acute, it is usually gradual (over 60 per cent of my patients); most of the patients in my series gave a history of disturbances of from one to more than nine years. The symptom most often complained of was pain, neuralgic in character, which was usually not localized to the cutaneous distribution of one or several spinal roots. The more diffuse character of the root pain is one of the features which distinguishes adhesive arachnoiditis from tumor of the spinal cord. As in nerve root irritation from other causes, the pain was often aggravated by movements of the spine or by forced expiratory movements such as straining, sneezing, or coughing.

In some patients motor disturbances and in others sensory symptoms were the first to occur. The motor disturbances consisted of weakness with or without spasticity in one or more limbs, so that, unless the lesion was below the level of the conus and cauda equina, spastic paraplegia was more or less well marked. Especially when the lumbar region was affected, there was weakness with wasting of the involved muscles with diminution or loss of tendon reflexes.

The sensory disturbances involved the dermatomes below the level, but the upper level of cutaneous diminution or loss was often not well defined, and if the patients were examined at intervals, it was not rare to find a gradual ascension of the upper level of sensory disturbance. In some patients the sensory diminution or loss was greater on one side of the body than on the other side, and in others the disturbances involved the two sides equally.

There was dissociation of cutaneous sensibility disturbances—preservation or very slight involvement of tactile with marked diminution or loss of pain and temperature senses in almost one-third of the patients. Muscle-joint-tendon and vibratory sensibilities were involved in many cases, but when the clinical picture was mainly that of an intramedullary lesion, the deep sensibilities were not disturbed.

As is encountered so often in intrinsic disease of the spinal cord, patchy areas of dysesthesia or hyperesthesia were sometimes found. Bladder and rectal disturbances occurred very often, especially when the signs pointed to involvement of the lumbosacral cord and roots of the cauda equina.

Lumbar puncture, manometric tests, and examination of the cerebrospinal fluid will yield valuable information regarding the question whether one is dealing with an intrinsic cord disease or whether there is also an adhesive process in the subarachnoid space. Occasionally fluid cannot be obtained by puncture; in the majority of cases fluid is found, but the tests for the patency of the spinal subarachnoid space demonstrate that there is partial or complete subarachnoid block. The fluid may be xanthochromic but usually it is clear, and in almost one-half of my patients the cell count and total protein were within normal limits. In the other cases some abnormality was found in the fluid: either an increase of cells, which was rare; or an increase of total protein and a corresponding increase of globulin, which was frequent. The highest total protein values I have seen have been between 200 and 500 mg. per 100 cc. of fluid.

A lipiodol examination may demonstrate not only a spinal subarachnoid block but sometimes the oil may be arrested, not in its entirety—as usually occurs in spinal compression by tumor—but in droplets over a considerable extent of the canal both above and below the suspected site of the lesion (Vincent, 1930; Guillain, 1932; Paulian and Turnesco, 1933; Marinesco, 1934; and others). In a number of my patients, the oil was arrested at a single level which was found at operation to be the site of marked adhesions, while in a smaller number a diffuse arrest was observed (Fig. 84).

DIAGNOSIS

From the data given, it is clear that in most instances the clinical picture is usually that of a spinal cord affection which has many of the features of intrinsic cord disease, but in which manometric tests demonstrate a more or less well-marked block in the spinal subarachnoid space with or without increase of total protein and of globulin. If the cord is compressed by a localized collection of fluid, the symptoms may simulate compression from some other cause and the question of spinal cord tumor is naturally raised. Most writers on the subject declare that in most instances it is difficult, if not impossible, to differentiate between a localized adhesive process in the meninges and a cord tumor, but this has rarely been the case in my personal experience.

In the first place, the development of symptoms and signs of disturbed function is seldom as regular as in compression of the cord by tumor. If one analyzes the histories given by patients in whom an adhesive process in the arachnoid is found at operation, one is impressed by the fact that the story is usually that of a more diffuse interference with spinal cord functions. The duration of disturbances is very often much longer than is seen in most cord tumors. There are usually findings which do not fit in the usual picture of spinal cord tumor: root pain may not be at or near the upper level of the lesion, and the successive involvement of motor and sensory functions with upward shifting of the sensory level is not that which is usually observed in spinal cord tumors. From all of the facts, one should be led to conclude that if the case is one of tumor, it is a case with an unusual course. That a subarachnoid block is demonstrated by manometric tests or by lipiodol does not make the suspicion of compression by neoplasm any greater, for it is well known that adhesive processes in the arachnoid are very frequent in a variety of intramedullary affections.

It is a more difficult matter to distinguish between an intrinsic cord disease with diffuse secondary adhesions in the leptomeninges (in which little or nothing can be accomplished by surgery) and a secondary or possibly primary adhesive arachnoiditis in which the cord is locally pressed upon by a cystic collection if fluid or by a thick band of adhesions (in which improvement may be marked after release of encysted fluid or division of contricting bands). Whatever may be the cause for the leptomeningeal adhesions, it cannot be denied that the adhesions may disturb the functions of the spinal cord and nerve roots by direct pressure, and by interference with the vascular supply of the cord aggravate a pre-existing intramedullary lesion. Therefore, in the present state of knowledge, exploratory laminectomy is generally indicated.

Treatment

When the upper level of the lesion has been ascertained, and the membranes exposed by laminectomy, the dura should be incised with great care because the spinal cord and leptomeninges may be adherent to the inner surface of the dura and the cord easily injured. After the edges of the dural incision have been retracted, fine adhesions should be carefully separated and a thick band which constricts the cord be looked for and, if found, divided. If there are adhesions which appear to press upon the posterior spinal vessels, they must be carefully divided without injury to the vascular structures. If there is a leptomeningeal cyst which compresses the cord, it should be incised and, if possible, some of the cyst wall excised.

If the adhesions are found to extend beyond the limits of the wound, the arches of one or two more vertebrae should be removed in the effort to expose all of the adhesions. If the lesions extend beyond this, it is probable that the disease is a diffuse one which cannot be relieved by surgery. A free flow of fluid from the cephalic part of the subarachnoid space after adhesions have been divided, is always a satisfactory occurrence as it indicates that there are few, if any, adhesions above the exposed part of the cord.

The adhesions are most often found on the posterior and lateral aspects of the spinal cord and especial attention must be paid to the emerging nerve roots, and any bands constricting them must be divided.

Prognosis and Results

The outlook after operation will depend to a considerable extent upon the intramedullary changes that have occurred. If the adhesive process is well localized and the symptoms have not been of more than one to two years' duration, the patient may be relieved of all or almost all disturbances by the operation. The longer the symptoms have existed, the greater the probability of irremediable cord changes and the poorer the outlook for relief. If the adhesive process extends over a great part of the spinal leptomeninges, the outlook for improvement is small. However, some of these patients may be improved if, by good fortune or good judgment, a part of the cord which has been most compromised by the adhesive process has been exposed, and adhesions which compressed the cord have been divided or the contents of a cyst evacuated.

Therefore, the results which can be expected after operative intervention will depend upon a number of factors: the duration of the meningeal adhesive process, the degree of intrinsic cord change, the degree to which the symptoms were due to compression of the cord. As the cases reported in the literature of the subject have seldom been analyzed from these points of view, it is difficult to arrive at a conclusion regarding the results of operative therapy. From the analysis of my personal series of 38 cases, the following conclusions were arrived at: In general, the prospect for great relief is small, because the patients have been operated upon at a late stage; in more than one-half of my patients, little or no improvement followed the surgical intervention. In about 25 per cent of the patients, in whom the adhesive process appeared localized with or without compression of the cord by bands, and in whom the symptoms were of less than two years' duration, there was considerable improvement in the spastic paraplegia and the sensory disturbances, so that bedridden individuals were able to be up and about again; bladder disturbances, when they existed, were not relieved, and the patient remained an invalid. Finally, in about 25 per cent of the patients the relief was complete or almost complete; most of the symptoms and signs disappeared, so that the individual was able to return to his work and usefulness. In the majority of these patients who were followed for many years, the relief was permanent; in a small number, the symptoms again became aggravated after one or several years.

The improvement after operation may occur rapidly, as in a patient with marked level signs, who was free of most disturbances after three months; or the improvement may be slow over the course of several years.

ABSCESS OF THE SPINAL CORD

Suppuration within the substance of the spinal cord is a relatively rare condition. Up to the year 1926 only 29 cases had been reported in the literature of the subject (Woltman and Adson, 1926), and up to the present time, less than 10 additional cases have been published. Personally, I have never operated upon a patient with an abscess in the substance of the spinal cord.

A study of the cases from the literature shows that infections or suppurative processes which may cause metastatic intracranial suppuration may also cause a purulent process within the substance of the spinal cord, and it is probable that the rarity of the latter as compared to the former, is mainly due to the relatively smaller total substance of the spinal cord. Pulmonary suppuration, especially in bronchiectasis, is a frequent cause, as are malignant endocarditis, infections in the genito-urinary tract, purulent processes in the neighborhood, such as diseases of the spine (Pott's disease), infected dermoid or meningocele, and meningitis. In some patients exposure to cold, and in others a trauma, preceded the symptoms and signs of the abscess; in a few patients the intramedullary suppuration occurred without any known cause.

Abscesses of the spinal cord have occurred more often in males than in females, and have been encountered at all ages from infancy to old age. The abscess may be small and localized or large and extending for more or less of the entire length of the spinal cord. In most of the cases collected by Woltman and Adson, the location and extent of the abscess were ascertained at a postmortem examination. In four of the cases, there were multiple abscesses and in the remainder there was a single abscess which was either small and involved one cord segment or less, or large and extended over a number of cord segments. In almost one-half of the cases the abscess extended over more than eight cord segments, and in five it extended over the greater part of the spinal cord. In some instances, the entire central canal was filled with pus, while in several the abscess extended into the medulla or pons. In four of the patients, one abscess or multiple abscesses of the brain also existed.

The collection of pus may be limited to one-half of the spinal cord or both sides may be involved. The abscess is most apt to be situated in or near the posterior gray horns or in the central gray substance, but it may involve the greater part of the diameter of the cord; it is always surrounded by an area of myelitis. Inflammatory changes and secondary degeneration may be found in many cord segments both above and below the actual location of the abscess. In almost one-half of the patients there was also a meningitis with or without organisms in the spinal fluid. In this connection, however, it must be remembered that most of the reports of the lesions were of those found at autopsy and that therefore in many cases the meningitis might have been a terminal meningeal infection.

The earliest reports on abscess of the spinal cord are those of Hart, 1830 (Woltman and Adson) and two cases of Ollivier, 1837 (Hitchcock). In both cases of Ollivier, the lesion followed a vertebral trauma.

The symptoms of spinal cord abscess usually develop rapidly, with sudden or very rapidly progressive loss of power in the affected limbs and with the sensory and other disturbances characteristic of a transverse lesion.

As the lesion is within the substance of the spinal cord, one might expect that dissociation of cutaneous sensibilities—preservation of tactile and loss of pain and temperature sensibility—would often be found, but the advance of the disease is usually so rapid that sensory dissociation is unusual. However, in a few of the published case reports, the progress of motor and sensory symptoms was less rapid, one limb after another was involved with hyperactive reflexes and with dissociated disturbances of cutaneous sensibility. Usually the advance of loss of power and of sensation progressed so rapidly that in a few days there were the signs of a complete transverse lesion of the spinal cord, with loss of all cutaneous and tendon reflexes and of control of the functions of the bladder and bowels.

The spinal cord disturbances may begin with pain and cutaneous hyperesthesia followed in a few days by loss of power and of sensation; or numbness of the limbs may be the first symptom. Even after the signs of a complete transverse lesion are evident, there may be an upward ascension of the level of anesthesia and analgesia.

The symptoms are generally those of a rapidly advancing intrinsic cord lesion which often begins with fever and sometimes with rigors. In most of the patients the course is very short; after a few days, motor and sensory loss are complete and the limbs are flaccid. If life is sufficiently prolonged, signs of meningitis appear and death occurs from a terminal lesion such as bronchopneumonia. In more than one-half of the collected cases, there was a flaccid paralysis with loss of all reflexes when the patients first came under observation.

The spinal cord symptoms due to the abscess must be distinguished from those due to the primary lesion. Thus, the motor and sensory loss due to fracture of the spine may be immediate, but the symptoms due to the suppuration within the substance of the cord may appear later (cases of Ollivier, 1837; Feinberg, 1876; Cavazzani, 1899). The same statement is true when the primary disease is situated in the vertebrae (Pott's disease—cases of Schlesinger, 1897; Turner and Collier, 1904; metastatic carcinoma of vertebrae—cases of Turner and Collier, 1904), and when the cord abscess is secondary to suppuration around or in the vertebral canal (infected dermoid—case of Dubreville, 1887; ruptured meningocele at birth—Wolff, 1909).

When, in addition to the spinal cord lesion, there is suppuration within the cranial cavity, the first loss of power and sensation may be caused by the brain abscess (cases of Nothnagel, 1884; Homen, 1895; Chiari, 1900; and others).

As a rule, the course of a spinal abscess is very rapid and the disease is a most serious one. The clinical picture is that of acute transverse myelitis, and that diagnosis was made in a number of the cases. The rapidity with which the symptoms and signs develop is like that met with in many cases of extradural spinal abscess and in patients with primary or metastatic malignant disease of the vertebrae. If the patient is seen only when the signs of meningitis are advanced with characteristic changes in the spinal fluid withdrawn by lumbar puncture, the differentiation from the transverse cord lesion that occurs from meningomyelitis may be impossible. However, in those conditions in which suppuration in the central nervous system is a relatively frequent complication—pulmonary suppuration and bronchiectasis, tubercular disease of the lungs or of the vertebrae—the possibility of an abscess of the spinal cord must be kept in mind. It was this viewpoint, which made it possible for Nothnagel (1884) and Homen (1895) to make the correct diagnosis.

In three patients an exploratory laminectomy was performed on account of spinal cord symptoms. In the case of Cavazzani, the abscess was found and the pus evacuated, but the patient remained totally paralyzed below the level of the lesion. In the case of Hitchcock (1917) in which the spinal cord disturbances developed during the course of three weeks, "the clinical history seemed to point to an infection of the cord, which had at first encroached on the posterior columns and then involved a transverse myelitis at the seventh dorsal segment," and a laminectomy was performed, but the abscess was not found because the cord was not aspirated.

In the case of Woltman and Adson (1923), the patient had had an attack of myelitis two years earlier and the progress of the symptoms had been gradual. The patient was seen when she had a spastic paraplegia, and manometric tests demonstrated a spinal subarachnoid block and yellow fluid. A diagnosis of meningomyelitis with spinal block was made and an exploratory laminectomy was performed. The cord was found to be greatly swollen and, upon incision, pus was evacuated from an encapsulated abscess about 1.5 cm. in length. The result of the operation was brilliant and the patient made a remarkable and almost complete recovery.

All of these facts show that there is no reason why an abscess of the spinal cord cannot be satisfactorily treated by the surgeon. Spinal cord abscesses are rare, but if the diagnosis is *made* or *suspected*, an exploratory laminectomy should be performed, the cord should be aspirated or incised, and the pus evacuated.

RADICULITIS OF THE CAUDA EQUINA

In 1914, Kennedy and Elsberg described a progressive inflammatory disease of the roots of the cauda equina, which presented a clinical picture similar to that of cauda equina tumor. Single cases had been previously described by Oppenheim, Balint and Benedikt, Raymond, and others.

An experience extending over many years has shown that in some of the cases in which the radiculitis appeared to be the only lesion, the condition was really secondary to another process in the neighborhood. One of my patients, in whom an exploration of the cauda equina revealed only swollen and congested nerve roots, died several years later from a generalized sarcomatosis with a large extradural tumor in the lumbosacral region. In another case, the swollen roots were probably produced by a very advanced arthritis of the lumbar spine. Therefore, before one arrives at the conclusion that radiculitis of the cauda equina is a primary process, one must be certain that there is not some other explanation for the congestion and swelling of the caudal roots. However, because, in most of the patients, another lesion could not be discovered and because many of them recovered after an operation, it appears certain that primary radiculitis of the cauda equina does occur.

The etiology of the disease is uncertain. Some of our 22 patients gave a history of trauma to the back; in others there was a story of a preceding generalized infection, such as influenza; in others there had been a local infection, such as inflamed varicose veins of the lower limbs, gonorrhea, or endometritis. In none of the patients was there a history of preceding syphilitic infection, and in all of them, the Wassermann test was negative in both blood and spinal fluid. (Under the name neuritis of the cauda equina, Marek, in 1909, described an inflammatory disease in the horse, which followed trauma.)

Most of the patients were adults over 40 years of age, and the two sexes were about equally represented. In almost two-thirds of the patients, the history dated back more than one year.

All or only some of the roots may be altered in appearance. The affected roots appear to be swollen and congested. Adhesions between adjacent roots were found in only one case. In most of the patients there were no evidences of an adhesive process in the leptomeninges: the arachnoid had a normal appearance and the inner surface of the dura was smooth and glistening.

The disease usually begins with sharp shooting pain in the back or in the thigh and calf of one limb or both limbs. Tingling and numbness of parts of the affected limbs soon follows. This is soon followed by increasing weakness of the lower limbs, with weakness of dorsiflexion of the foot, often with dropped foot; sensory disturbances; and disorganization of the functions of the bladder and rectum.

The diminution of power in the lower limbs consisted mainly of weakness or loss of the ability to flex the feet dorsally and weakness or loss of power of extension of the legs at the knee. The Achilles reflexes and, in most of the patients, the knee jerks were lost.

The abdominal reflexes were always preserved.

Atrophy with fibrillations of the anterior tibial muscles and of the peronei was usual, and in the advanced cases there was considerable atrophy of the muscles of the thighs and of the gluteal musculature.

Pain, affecting one lower extremity, was complained of by 80 per cent of the patients. In almost one-half of these the pain remained confined to one side; in the others the pain was felt in both lower limbs. The pain was often felt on the posterior surface of one thigh or down the entire extremity, and not rarely the patients had been treated for supposed sciatica. In about one-fourth of the patients, there was also pain in the lower part of the back.

The objective sensory disturbances showed an astonishing uniformity; in all, the sacral roots were most affected. In some patients the sensory loss was limited to the sacral roots; in others several or many of the lumbar roots were also involved. If the lumbar roots were affected, the upper limit of sensory disturbance was often ill defined.

All in all, however, the sensory loss was more symmetrical in the two lower limbs than in tumors between the roots of the cauda equina.

As would be expected, the more markedly the lower sacral roots were involved, the more marked was the sphincter disturbance. The disorganization of the functions of the bladder and rectum was complete in most of the patients; incontinence of urine with dribbling, or constipation or incontinence of fluid feces, or both was the rule.

Lumbar puncture and manometric tests usually showed no evidence or only little evidence of subarachnoid block. The fluid was generally clear and colorless, although in two patients it was very slightly yellow. There was seldom any increase of total protein or globulin and only occasionally a slight increase of cells above the normal.

The roentgen examination of the spine, excepting for a moderate degree of arthritis in some of the patients and of a spina bifida occulta in one case, failed to show anything abnormal.

From this short description, it will be seen that the symptoms and signs of radiculitis of the cauda equina are similar to those of cauda equina tumor (*q.v.*), and in many of our cases, the diagnosis before operation was uncertain. An analysis of the symptoms and signs of a series of tumors of the spinal cord and of a series of cases of radiculitis was made by the author and Dr. Constable. Roentgenographic evidence of enlargement of the spinal canal makes the diagnosis of tumor certain, but when there are no definite changes of the bones, the patient may have either a tumor or a radiculitis. Absence of increase of protein in the spinal fluid and evidence that the subarachnoid space is patent, favor a diagnosis of an inflammatory lesion.

In the majority of instances, the diagnosis is uncertain and an exploratory laminectomy will have to be performed. At the operation, the difference between the color and size of the affected roots and the appearance of the normal roots is striking and easily recognized. If the only lesion that can be found is a swelling of caudal roots, the affected roots should be sponged off with weak tincture of iodine (2 per cent) or with 1-2000 bichloride solution.

All of the patients in whom a laminectomy was performed, recovered from the operation. In a few cases the postoperative improvement was rapid and began soon after the laminectomy, but in the majority the improvement was very slow and several years elapsed before power and sensation were regained. Recovery was seldom complete and some weakness at the ankle and sensory disturbances remained. Fortunately, complete restoration of the functions of bladder and rectum was almost the rule. Several of the patients died after months or years from some other cause; and, as already mentioned, in some cases the course after operation showed that the swelling and congestion of the caudal roots was entirely secondary to some other disease. In these patients, there was no improvement after the operation.

CHAPTER VII

TUMORS OF THE SPINAL CORD, NERVE ROOTS, AND MEMBRANES*

I. FREQUENCY AND INCIDENCE

THE RESULTS OF THE OPERATIVE TREATMENT OF TUMORS OF THE SPINAL CORD AND MEMBRANES ARE AS BRILLIANT AS ANY OBTAINED in the entire realm of surgical therapy, and the importance of early diagnosis is therefore not to be underestimated. Although frequent reference will be made to the literature of the subject (which is large), the statements made in Chapters VII to XIII are based mainly upon personal experiences.

Little is known regarding etiology. There is no convincing evidence that there is a connection between trauma and the development of a spinal neoplasm. Heredity does not seem to play a part, although isolated instances of spinal tumors in several members of one family have been reported. Thus, I have operated upon an aunt and her niece for cord tumor. Pregnancy and labor have been mentioned by many authors as having preceded the rapid appearance of spinal symptoms; and in about 5 per cent of the married females in my series, the symptoms of a spinal cord tumor either began or became markedly aggravated after confinement.

INCIDENCE

Of the 275† cases of my series, 122 were in males and 153 were in females. This predominance of females over males is due to the relative frequency of the meningiomas in women.

In the collected cases of Stursberg (1908) there were 80 males and 36 females; while Steinke (1918) found 169 cases in males as

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* For remarks on multiple tumors, see Appendix, page 555 *et seq.*

† Actually there were 296 patients, but the records of 21 were not complete.

TABLE V

RELATIVE FREQUENCEY OF SPINAL TUMORS IN MALES AND FEMALES

Tumors	*Male*	*Female*	*Totals*
Extramedullary	64	111 (63%)	175
Extradural	48	33	81
Intramedullary	10	9	19
Totals	122	153 (55%)	275

compared with 136 in females. On the other hand, of the cases of tumor of the spinal cord reported at the Ninth Congress of the International Society of Surgery in 1932 (Robinson, Denk, Goldstein, Jerasik, Dominici) the two sexes were equally represented.

AGE

Spinal cord tumors may occur at any age, but they are especially likely to occur in adult life between the twenty-first and the sixtieth year of age. The following figures are based upon the author's series:

Under 10 years	5 cases	
11–20 years	28 cases	79 per cent
21–30 years	41 cases	
31–40 years	60 cases	
41–50 years	70 cases	
51–60 years	47 cases	
61–70+ years	24 cases	

The relative frequency of different histological types may be seen from the following table of the cases of my series. Cases of multiple tumors, as occur in myeloma, chordoma, and Hodgkin's disease are not included in the table.

SIZE

The majority of cord tumors are small, measuring from 3 to 8 cm. in length and from 2 to 4 cm. in thickness, but larger growths are often encountered. Growths in the lumbosacral part of the canal, surrounding the conus and roots of the cauda equina are frequently very large, filling up a much enlarged spinal canal. The largest tumor I have met with in this region was 15 cm. in length. Because the typical cauda equina tumors are so large, I have called them "giant" tumors. Intramedullary growths may be large, extending over a considerable distance up and down the spinal cord.

Primary or secondary growths outside of the dura may be fairly well localized under several vertebral arches, or they may extend up and down the vertebral canal for considerable distances. They often surround the dural sac.

Metastatic extradural tumors may extend over large areas of the vertebral canal, as may the tumor masses outside of the dural sac that occur in Hodgkin's disease and in multiple myeloma. In the so-called dumbbell growths, the extravertebral part of the neoplasm may be very large, as in the cases of cervical neurofibroma of Flatau and Sawicki, and in the vertebral chordomas.

TABLE VI

RELATIVE FREQUENCY OF HISTOLOGICAL TYPES

Types of Tumors	*Extradural*	*Intradural*	*Extradural and Intradural*	*Intramedullary*	*Totals*
Perineurial fibroma	5	54	—	—	59
Meningeal fibroblastoma	4	69	—	—	73
Sarcoma	22	9	—	—	31
Angioma Hemangioblastoma	4	5	—	—	9
Fibroma	4	2	—	—	6
Neurofibroma (von Recklinghausen)	1	3	1	—	5
Dermoid	0	4	—	—	4
Lipoma	1	2	—	3	6
Ependymoma	0	0	—	11	11
Cyst	2	1	—	—	3
Ganglioneuroma	2	—	—	—	2
Chondroma Herniation of nucleus pulposus	27	—	—	—	27
Granuloma	2	—	—	—	2
Metastatic and secondary	11	2	1	—	14
Unclassified	—	8*	—	—	8
Glioma	—	—	—	14	14
Tuberculoma	—	—	—	1	1
Totals	85	159	2	29	275

* Mostly tumors of the conus and cauda equina, many of which, perhaps, should be classified as intramedullary growths.

VERTEBRAL LEVELS

In my experience the growths were found at the following vertebral levels:

TABLE VII

UPPER VERTEBRAL LEVELS OF 296 TUMORS OF THE SPINAL CORD

Vertebral Level	*Extradural*	*Intradural*	*Extradural and Intradural*	*Intramedullary*	*Totals*
Cervical 1 to 3	4	12			16
Cervical 4 to 8	17	28	2	5	52
Thoracic 1 to 3	10	24	1	5	40
Thoracic 4 to 8	25	48		1	74
Thoracic 9 to 12	12	36	1	3	52
Lumbar 1 to 3	6	20		15	41
Lumbar 4 to 5	8	12			20
Sacral	1				1
Totals	83	180	4	29	296

Tables VII and VIII show that the relative frequency at the different vertebral levels corresponds to the relative number of cervical, thoracic, and lumbar vertebrae, and also to the relative length of the cervical, thoracic, and lumbar parts of the spinal cord, as given by Ravenel (1877).

TABLE VIII

FREQUENCY OF TUMORS COMPARED TO NUMBER OF VERTEBRAE AND TO RELATIVE LENGTHS OF THE CERVICAL, THORACIC, AND LUMBAR CORD

Vertebrae	*Length of Spinal Cord (Ravenel)*	*Tumors*
Cervical 7 = 29%	10 cm. = 23%	68 = 23%
Thoracic 12 = 50%	26 cm. = 58%	166 = 56%
Lumbar 5 = 21%	8.5 cm. = 19%	62 = 21%

These figures demonstrate that, in general, the relative frequency of spinal cord tumors corresponds to the relative lengths of the different parts of the cord and to the relative number of cervical, thoracic, and lumbar vertebrae. On page 227, we shall indicate that the meningeal fibroblastomas do not follow this rule.

The sites of predilection of spinal growths vary to some extent with the pathological type. The lipomas are most often encountered in the cervical and the lumbar regions; the meningiomas are most frequent in the thoracic region and especially between the second and the eleventh thoracic vertebrae; extradural cysts occur most often between the sixth and the tenth thoracic vertebrae; herniations of a nucleus pulposus are most often derived from the lumbar intervertebral cartilages.

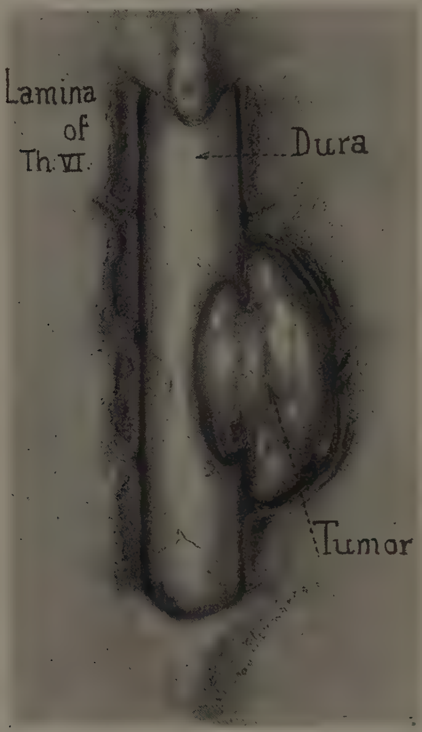

FIG. 97. An extradural chondroma which has extended into the spinal canal through a foramen between two vertebrae.

RELATION OF TUMORS TO THE CORD AND MEMBRANES

Spinal tumors may originate (1) from the spinal cord, nerve roots or membranes, or (2) from the soft tissues or bone. The growths that begin from the latter may primarily or secondarily press upon or interfere with the circulation of the cord.

Extradural tumors may arise from the dura, from nerve roots outside of that membrane; or from the fat, blood vessels, ligaments, or bone in the epidural space; or from the ligamentous or osseous tissues forming the walls of the vertebral canal.

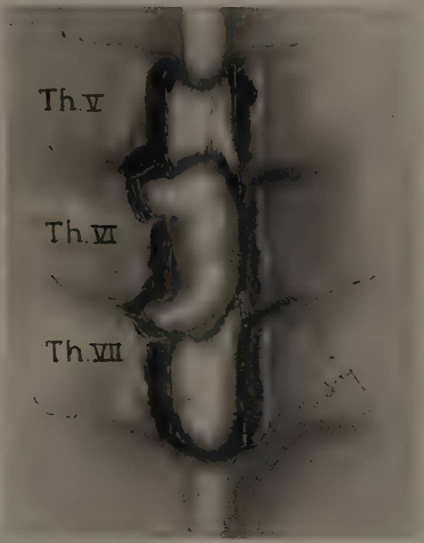

FIG. 98. An extradural lipoma which extended into the spinal canal through two intervertebral foramina.

In the classification of extradural new growths one must differentiate those that are *primarily* extradural from those that *secondarily* extend into the vertebral canal.

Primary extradural tumors are (1) those that arise from dura, nerve roots, epidural fat, and blood vessels, and are located entirely within the vertebral canal; and (2) those that arise from the walls of the vertebral canal—that is, from ligaments, intervertebral cartilages and bone—and from their inception lie entirely within the vertebral canal.

Secondary extradural tumors are growths that arise from structures outside of the vertebral canal and, secondarily, invade the extradural space.

Metastatic tumors may be either primary or secondary.

Growths which spring from nerve roots or nerves outside of the spinal canal, may extend into the spinal canal through an intervertebral foramen; and, vice versa, growths primarily inside of the canal may extend through one or more intervertebral foramina into the surrounding soft tissues. In many instances malignant disease of the soft tissues which surround the vertebrae or a growth in the thorax extends into the canal through the foramen between two vertebrae (Figs. 97, 98). Thus there may be formed the so-called dumbbell tumors which are illustrated in Fig. 99, examples of which have been reported by Börner, 1903; Elsberg, 1925; Borchardt, 1926; Heuer, 1929; Naffziger and Brown, 1933; Antoni, 1936; and others. In the cases called cervical neurofibroma by Flatau and Sawicki (1922), the extradural growth is derived from cervical roots, grows outward, and forms a large mass in the neck. I have observed several instances of this type of growth (Fig. 100).

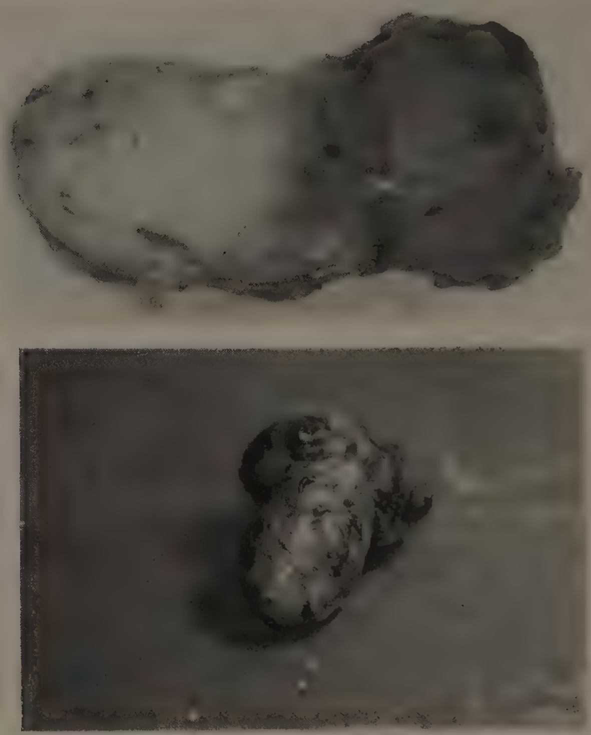

FIG. 99. Two extradural and intradural, so-called dumbbell-shaped, tumors.

Cases of dumbbell-shaped tumors have been reported by Schultze, (1903, 1912), Bing and Bircher (1909), and by others, in which one part of the tumor was outside and the other part inside of the dura. Antoni (1920) collected 23 such cases, including several of his own.

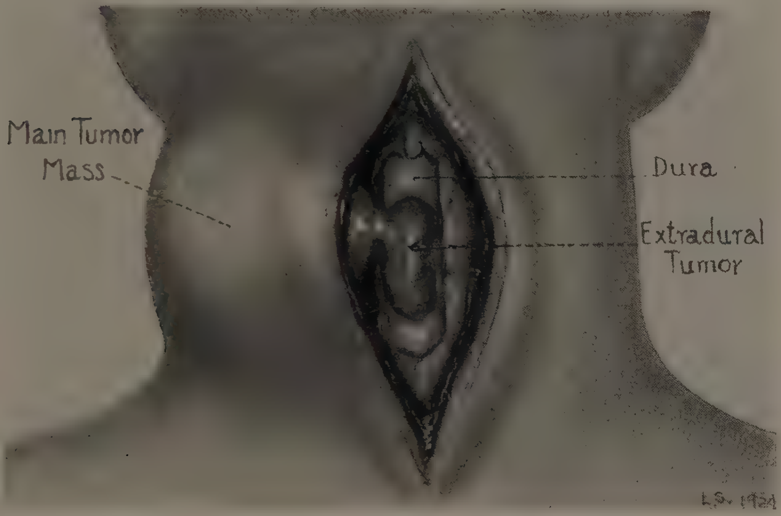

FIG. 100. An example of so-called cervical neurofibroma.

Congenital cysts are most often extradural, but as they are derived from herniations of the arachnoid through the opening in the dura for a nerve root, they may be both intra- and extradural or entirely intradural. Finally, a tumor may originate in an intervertebral foramen—perhaps from a nerve root or ganglion—and grow outward, forming an extraspinal mass; or it may grow inward through an opening in the dura and form an intradural tumor.

Extradural growths, both primary and secondary, may lie posterior, lateral, or anterior to the dura; but it in many instances they surround the dural sac.

Intradural tumors are either extramedullary or intramedullary. It is difficult to state with certainty that a growth is primarily intramedullary, especially when it has developed from the inner surface of the pia mater of the cord, but subpial growths are very rare. Primary intradural tumors may grow through the opening in the dura from a nerve root and be both intra- and extradural, as in the case reported by Schroeder (1917) and in some of the cases reported by Antoni (1920).

RELATION OF INTRADURAL TUMORS TO THE LEPTOMENINGES

Extramedullary growths may lie inside or outside of the arachnoid; very rarely, they may be subpial. In the majority of instances the growth, even though it is adherent to the inner surface of the dura, is covered by arachnoid, and at the operation this relationship between tumor and arachnoid can be established.

In order to determine the exact relations of the tumor to the spinal cord, a definite classification of the growths with reference to the surfaces of the spinal cord is necessary. We have for many years grouped our tumor cases in the following way (Fig. 101).

All growths on the posterior aspect of the cord, whether in the median line or laterally *behind* the posterior nerve roots are classified as *posterior* or dorsal growths; those that lie on the lateral aspect of the cord *in front* of the posterior roots but behind the dentate ligament are called dorsolateral; those that lie laterally in front of the dentate ligament but *behind* the anterior roots, are ventrolateral; those that lie on the anterior aspect of the cord in the median line or laterally but *in front* of the anterior roots are called anterior or

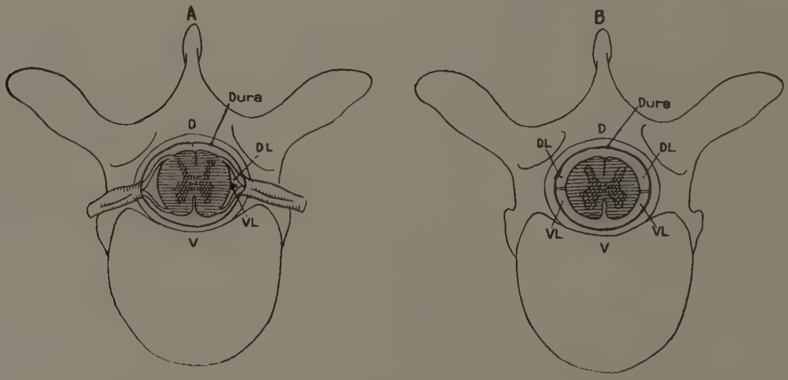

Fig. 101. The classification of tumors with reference to the surfaces of the spinal cord. A. At the level of the nerve root. B. At the level of attachment of a slip of the dentate ligament. *V,* ventral; *D,* dorsal; *VL,* ventrolateral; *DL,* dorsolateral. (Diagrammatic.)

ventral. This classification is useful, although growths may occupy several of these locations, or they may be *in front* of the cord but *behind* the anterior roots, or finally, a growth in one relation to structures may, as it increases in size, slip into some other relation to roots or dentate ligament. In my series of extramedullary spinal cord tumors, 60 per cent of the growths lay dorsal or dorsolateral and 40 per cent were ventral or ventrolateral. In a not inconsiderable number of instances, an extramedullary growth may develop away from the posterior spinal roots, and, in these cases, the growth may reach a relatively large size before there is any evidence of root pain.

Intramedullary growths may occupy any part of the cord; they are often found in the posterior or lateral parts of the cord and may appear on the lateral or dorsal surfaces. The growth often causes a fusiform enlargement at the affected level. With it is frequently associated syringomyelia with a cavity filled by yellow fluid. Kernohan, Woltman and Adson (1931) found coexisting syringomyelia, which extended for a considerable distance up and down the spinal cord, in 56 per cent of their cases in which an autopsy was performed.

While most intramedullary growths arise from the glia or nerve tissue proper, some growths undoubtedly arise from and in the more fibrous tissue of the posterior median septum.

THE MOBILITY OF THE SPINAL CORD AND THE MECHANICAL EFFECTS OF CORD TUMORS

The mobility of the cord, at various levels, has a great influence upon the symptoms and signs of an expanding lesion within the spinal canal. The actual possibility of movements of the cord in the dural sac, in an upward or downward direction (i.e., in the direction of the axis of the spinal canal) is very small. However, during movements of the vertebral column (especially during flexion), there is a slight change in the relation of segments of the cord to vertebrae; and the same is true during the act of breathing; especially during deep inspiration and in forced expiratory movements, such as occur during the acts of sneezing and coughing. If an individual flexes his vertebral column—as, for example, in the act of bending forward—each segment of the cord lies a little higher than it did before, and hence there is a slightly increased pull on the nerve roots. This pull is so small that it does not normally give rise to symptoms. When there is a tumor, however, the slight change that occurs is sufficient to cause an increased root irritation. This is one of the reasons why the affected part of the vertebral column is often rigidly fixed by the patient.

Both in our anatomical investigations and in our observations on the operating table, we have found that a cord segment is most movable in anteroposterior and lateral directions at and just below the level of each pair of spinal roots. Our attention was first called to this fact in a patient in whom we wrongly localized a growth on the posterior aspect of the cord on account of well-marked posterior column disturbances. At the operation, the tumor was found and removed from the anterior surface of the cord. It had pushed the cord backward so that the posterior columns were pressed against the dura and posterior bony wall of the vertebral canal.

On account of the mobility of the cord, the dislocation of segments of the cord by the direct pressure of a soft tumor may, as in the instance we have cited, cause confusing sensory disturbances.

There is another type of cord dislocation, however, which is not due to direct tumor pressure and the physics of which is not at all clear to us. We first began to investigate this subject some years ago when I operated upon a patient with a tumor on the lateral aspect of the cord, in whom motor disturbances first occurred on the contralateral side of the body, and in whom, at the time of our first and of succeeding examinations, the most advanced motor disturbances were on the opposite side of the body and the most marked sensory loss on the same side as the tumor. This "reversed" Brown-Séquard syndrome could be explained only on the basis of a greater involvement of the lateral columns of the cord on the side opposite to that on which the tumor lay, and an explanation of this condition of affairs had to be sought in the mechanical changes that might be produced by a growing spinal cord tumor. I have observed and operated upon a number of patients with this "reversed" Brown-Séquard picture. Some of these had extradural growths, and in others the tumor was intradural but firmly adherent to the inner surface of the dura.

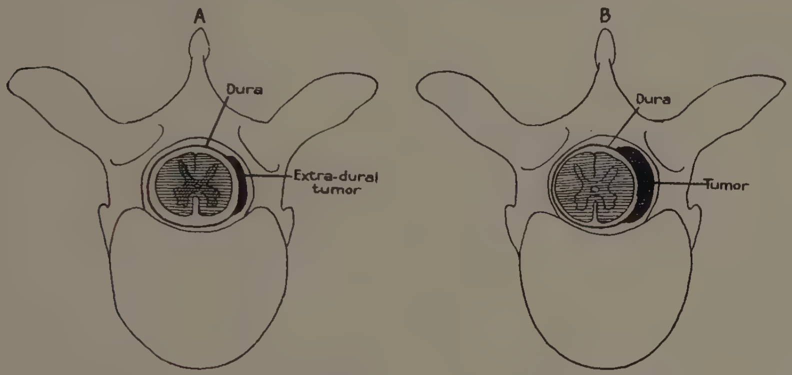

FIG. 102. The mechanical effect of an extradural tumor. A. When the growth is small; B, when the growth is increased in size. (Diagrammatic.) (For explanation see text.)

Although fully cognizant of the law of physics that, in a fluid medium, pressure is transmitted equally in all directions, I do not believe that this can be made to apply fully to the conditions which exist within the spinal canal where there is a fluid medium (the cerebrospinal fluid) and a more solid structure (the spinal cord) which is partly held in place by nerve roots and by the dentate ligament. In addition, it is important to remember that the fluid medium which surrounds the cord is more or less in motion and that, in general, there is a constant movement of fluid in a downward direction (caudad) on the posterior aspect and upward (cephalad) on the anterior surface of the spinal cord.

Without attempting to solve the physics if the process, I believe that the following is the only possible explanation for the changed position of the cord that I have seen at operations:

In Figure 102, A represents an extradural tumor on the left side of the dural sac in the earliest stages of its growth. When the growth has reached a certain size and before actual pressure upon the cord by the tumor has occurred, the cord will be found to have changed its position and to lie against the dura and the bony wall of the canal

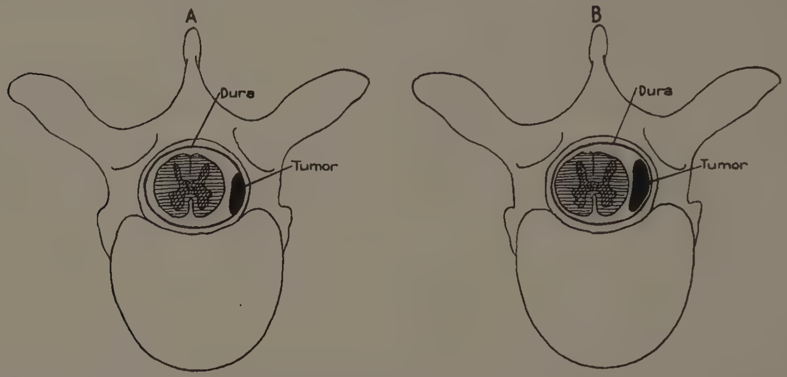

FIG. 103. The mechanical effects of an intradural tumor adherent to the inner surface of the dura (diagrammatic). (For explanation see text.)

on the side opposite to that of the tumor (Fig. 102 B). The actual physical condition that now exists is the following: the right side of the cord is in contact with a firm bony resistance, while the left side is more or less protected by a fluid water pad which distributes the pressure over that side of the cord. At this stage and as a natural result, the right side of the cord is more involved and symptoms referable to that side of the cord are more prominent.

A similar condition may obtain in the case of a growth which is firmly adherent to the inner surface of the dural sac and is not yet of large size (Fig. 103). If, on the other hand, the growth begins from the arachnoid, which is bound to the pia by numerous fine trabeculae, the cord is pushed to the side opposite that of the tumor only by the tumor itself when it has reached a certain size (Fig. 104).

The amount of dislocation of the cord varies considerably, therefore, with the location of the tumor and its attachments. The greatest amount of dislocation occurs in anterior and posterior growths, because the greatest range of mobility of the cord is forward and backward.

The dislocation may be so marked that the cord forms a regular knuckle backward, and the dorsal white columns of the cord are pressed firmly against the dura and the posterior wall of the spinal canal. Forward dislocation of the cord is also frequent. For obvious reasons, the amount of forward dislocation is not as easily appreciated.

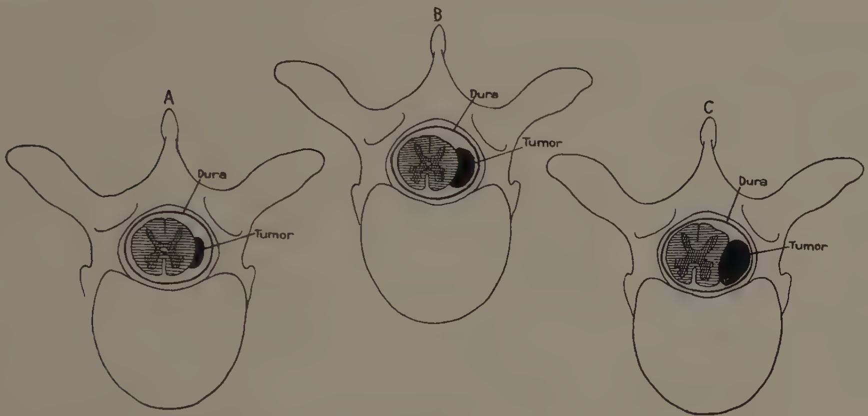

FIG. 104. The mechanical effects of an intradural spinal tumor not adherent to the dura (diagrammatic). (For explanation see text.)

Dislocation of the cord to one or the other side occurs to some extent in all lateral growths. As we have already seen, the lateral displacement may be very marked. It is clear, of course, that in most instances the cord is dislocated in several directions, backward or forward, and to one or the other side.

With this displacement of the cord, a certain amount of stretching of nerve roots on one or both sides occurs, and often causes bilateral root hyperesthesias.

Growths that develop from the inner surface of the arachnoid may be firmly fixed to the cord or may become adherent to it. Sometimes, however, they have a distinct mobility with respiration. I have seen instances in which the tumor moved up and down with respiration within the unopened arachnoid. Sargent refers to cases of this kind.

The amount of flattening and indentation of the cord depends upon the size of the growth and its consistency, and upon the relation of the tumor to the cord and the mobility of the cord in the affected region. As the amount of pressure upon the cord by a spinal tumor varies, so will the amount of the transverse diameter of the cord that is affected vary with the size of the growth and with certain other factors. There is, I believe, a grouping of fibers according to limbs and parts of limbs, not only in the posterior columns but also in the anterior and the lateral columns (i.e., in the pyramidal, spinothalamic, and other pathways). If only a part of the cord is affected by the increased pressure, then only a small part of a fiber tract may be compressed. If the pressure is from within outward, the fibers in each tract that are most mesially situated will first be subject to the increase of pressure. Vice versa, in a pressure upon the cord from the outside, it will be the fibers nearest the surface that will be first involved.

Much will depend upon the location of a tumor and the direction in which it is growing. Thus, for example, the pressure upon the cord by a tumor lying on its anterior surface near the midline may be very similar to that exerted by an intramedullary growth. It is well to remember that an anterior tumor may change the appearance of the cord at the affected level by pushing it backward, so that the cord looks broadened, as it does in some intramedullary growths. As a result, the motor and sensory disturbances and muscle atrophies from a tumor in front may be very similar to those from a growth within the substance of the cord. Similarly, the backward pressure of a growth in front of the cord may give rise to signs of disturbed function very similar to those produced by a tumor in the midline on the posterior aspect of the cord not in contact with sensory roots.

As an actual fact, the pressure is not always so gradual, and the patient is rarely seen at such an early period of the disease that it is possible to detect slight differences between, for example, a pressure backward from in front and a pressure forward from in back of the cord; in both instances the patient may be first seen at a time when the physical signs are similar to each other.

The cord may be flattened over a small or a large area, but the most advanced compression is seen in cases where there is a well localized deep depression in the cord tissue. The cord may be so much thinned that, to the naked eye, it appears as if there were no tissue between the ventral and dorsal pia mater.

The flattening may be more diffuse, so that over an area of one or more segments the cord has the appearance of a thin band, which, after the tumor has been removed, floats on a layer of fluid with a wavy motion due to the respiratory and cardiac pulsations of the cerebrospinal fluid.

Intradural tumors in the lowermost portion of the spinal canal, unless they originate directly from a nerve root or from the dura near the point of exit of a nerve root, make comparatively little pressure upon the caudal roots until they have attained a considerable size. They may push the roots in any or in all directions. Most often the roots are pushed backward or forward or to one or the other side, but it is not so rare for the roots to be separated so that some are displaced in one and others in another direction.

One of the earliest effects of an expanding lesion within the spinal canal is compression of the spinal veins and interference with the return venous circulation. Over the actual area of pressure all the vessels are usually small and empty, but below the compressed area, the veins will be found to be distended and more or less tortuous. The appearance of these veins is absolutely characteristic and extends over considerable of the cord below the affected level. From the appearance of these distended veins one can tell with certainty that there is some obstruction higher up if no tumor is found in the region exposed at operation. (Fig. 208)

As a spinal tumor increases in size, it interferes more and more with the circulation of spinal fluid in the arachnoid sac. When the downward flow of fluid is interfered with, the fluid is retained above the tumor and may exert a slight pressure on cord segments above the actual level of the lesion. When such a tumor is removed, the fluid escapes from above with a sudden gush.

A localized increase of intraspinal pressure may also interfere with the normal cardiac and respiratory pulsation of the cord. The pulsations of the cord are especially noticeable in the cervical region, and absence of pulsation just below this level is very significant of pressure at a higher level, a fact to which Oppenheim has also called attention.

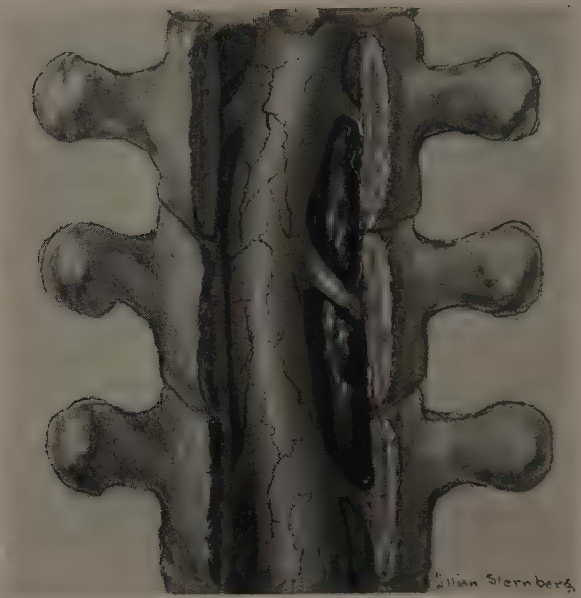

FIG. 105. An extradural tumor fixed in place by a spinal nerve root.

The degree of compression and the amount of dislocation of structures are not by any means always in proportion to the size of the growth, because a great deal depends upon the consistency and the location of the expanding lesion. The first effect of an intradural extramedullary growth is expended upon the tissue from which the growth originated. If the tumor started from a nerve root or its covering, the root becomes more or less stretched and flattened. A nerve root may offer considerable resistance to the advance of the growth and may fix the latter firmly against the dura or against the cord itself (Figs. 105, 106). If the growth is of firm consistency (e.g., meningioma) it is more apt to cause a deep depression in the cord than when it is soft or cystic, as in the perineurial fibroblastomas. The dentate ligament, especially the slips of the ligament attached to the inner surface of the dura, may offer considerable resistance to the forward or backward extension of a growth; and in many instances, a slip of the dentate ligament holds a tumor firmly in one position against the dura or against the cord.

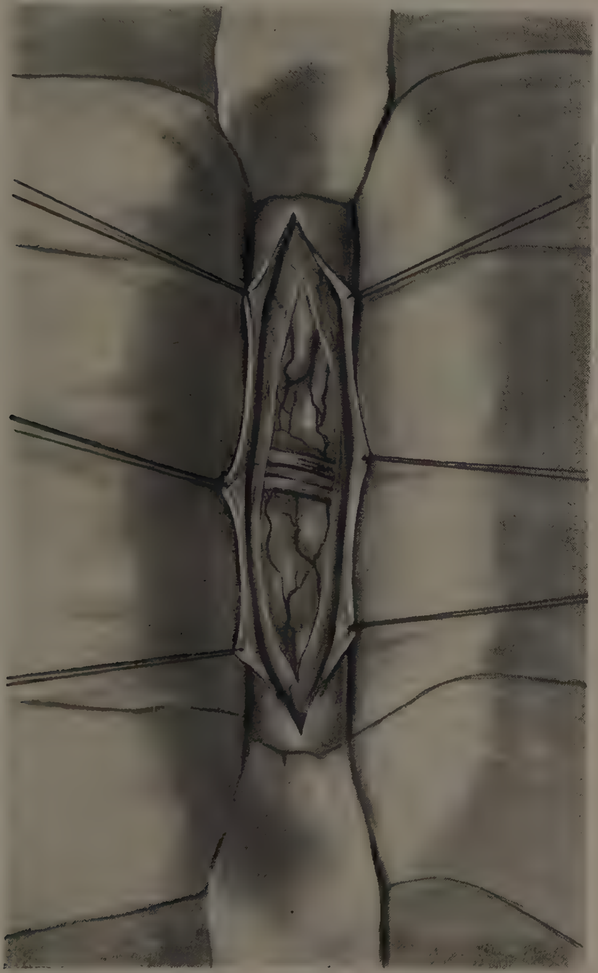

FIG. 106. A large tumor of the cord over which two bundles of a nerve root are tightly stretched. (Elaborated from sketch made at operation.)

THE GROSS STRUCTURE OF TUMORS OF THE SPINAL CORD

Meningiomas. There are many variations in the gross structure of tumors of the spinal cord, and there is no agreement regarding histological classification. The growths that, following Mallory and Penfield, we call meningeal fibroblastomas, are mesodermal growths which reproduce to varying degrees the structure of pacchionian granulations. They have been given a variety of names: dural endothelioma, psammoma (Virchow), sarcoma angiolithique (Cornil and Ranvier), meningeal sarcoma (Spiller), arachnoid fibroblastoma (Mallory), meningeal fibroblastoma (Penfield), meningoblastoma (Roussy and Oberling), meningioma (Cushing). The gross appearance of these growths is a characteristic one. They are usually rounded in shape (Fig. 107), well encapsulated, and have no tendency to invade the nervous tissue. They are usually firm growths, but if they have undergone degeneration, the vascularity of the surrounding soft tissues and bone is generally, though not regularly, increased. They are usually adherent to the dura and are most often subarachnoid although they may be found outside or inside the dura or arachnoid. The growths usually produce a more or less discrete depression in the spinal cord at the affected level (Fig. 108 A) and markedly angulate the cord (Fig. 108 A). The meningiomas occur more often in the female sex (80 per cent of the meningiomas of my

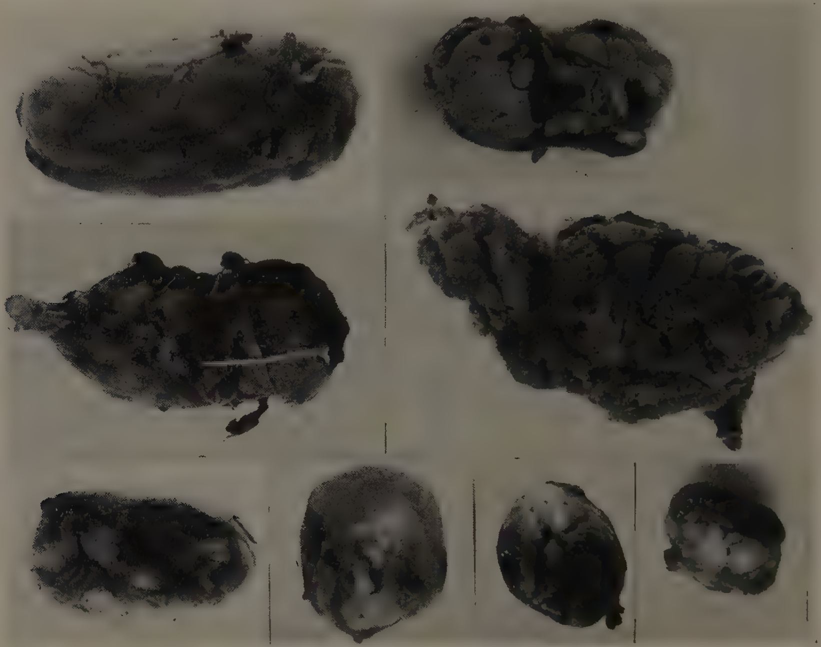

FIG. 107. Photographs of a series of spinal meningiomas.

series), and between the levels of the second to eleventh thoracic segments of the spinal cord, as seen in Table IX.

The incidence of meningiomas in the thoracic region is much higher than should be expected from the relative length of the thoracic spinal cord. The frequency of meningeal growths in the thoracic region is probably related to the differences in development of the membranes in the thoracic region as compared with other regions of the spine.

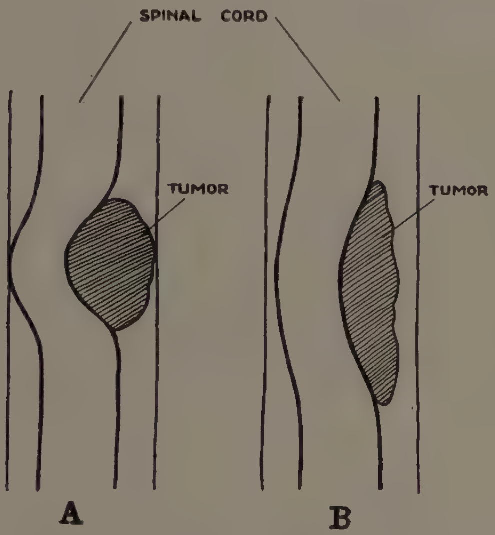

FIG. 108. The effects upon the cord of (A) a mengioma, and (B) a perineural fibroblastoma. (Diagrammatic.) (For explanation see text.)

Perineurial Fibroblastomas. There has been much difference of opinion regarding the origin of these growths; that is, whether they are formed from connective tissue or from the cells of Schwann. This is the explanation for the bewildering variation in nomenclature: fibroma, neurofibroma, neurinoma (Verocay, Antoni), perineurial fibroblastoma (Mallory, Penfield), schwannoma (Masson), peripheral glioma (l'Hermite, Leroux), fibroglioma (French authors), and so on. Our preference is for the term perineurial fibroblastoma for the solitary growths, and for the term neurofibroma for the growths that occur as a part of a generalized neurofibromatosis (von Recklinghausen's disease). Penfield has shown that

TABLE IX

FREQUENCY OF MENINGIOMAS AT VARIOUS LEVELS OF THE SPINAL CORD

		Number of Cases	
Cervical	1	2	
	2	1	
	3	1	
	4	0	
	5	1	
	6	2	
	7	1	
	8	2	
Thoracic	1	2	
	2	5	
	3	6	
	4	8	
	5	5	
	6	5	70%
	7	7	
	8	6	
	9	7	
	10	6	
	11	1	
	12	1	
Lumbar	1	2	
	2	1	
	3	1	
	4	0	
	5	0	

the latter differ histologically from the solitary tumors; in the solitary growths nerve fibers are not found in the tumor tissue either by nerve fiber or myelin stains, while in the tumors of von Recklinghausen's disease, nerve fibers can be demonstrated within the substance of the tumor.

The spinal perineurial fibroblastomas are usually soft in consistency and often contain cysts; they are attached to a posterior root and are rarely adherent to the dura. On the average, they are larger in size than the meningiomas and they vary greatly in size and shape (Fig. 109). In rare instances the growths extend through the opening in the dura from a nerve root, so that there is both an extradural and an intradural tumor. All in all, these growths do not make as deep a depression in the cord (Fig. 108 B) as the meningiomas, mainly because they are softer in consistency.

As compared with the meningiomas, which occur relatively often in the female sex after the fortieth year of life and in the thoracic part of the spinal cord, the perineurial fibroblastomas occur with almost equal frequency before and after the fortieth year of life, and almost equally often in the two sexes. If the relative number of cervical, thoracic, and lumbar vertebrae is taken into account, the tumors occur almost equally often at cervical, thoracic, and lumbar levels.

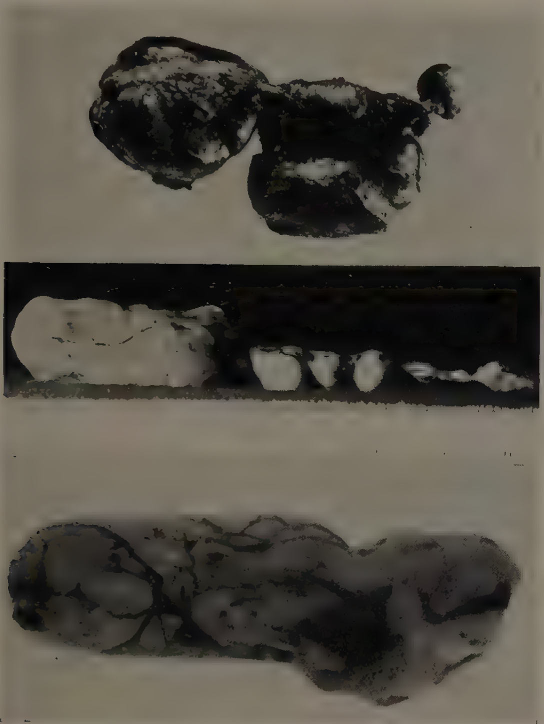

FIG. 109. Photographs of three spinal perineurial fibroblastomas.

The perineurial fibroblastomas occur both extradurally and intradurally, but are far more frequent within the dural sac.

Angiomas or *hemangioblastomas* occur as localized encapsulated growths, but are relatively rare (3 per cent of our cases). They have been encountered both extradurally and intradurally and have often been classified as vascular meningiomas, or angiolipomas, but true extramedullary angiomas do occur as in the case illustrated in Figure 110. They must be distinguished from masses of dilated blood vessels, congenital arteriovenous aneurisms (Elsberg, 1916; Sargent, 1925), and anomalous dilatations and varicosities of the spinal veins (*q.v.*).

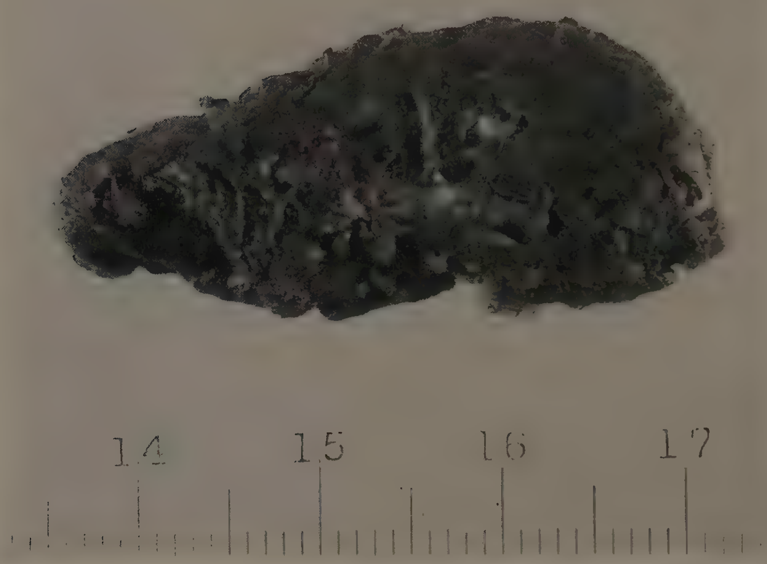

FIG. 110. An extradural angioma.

Lipomas are most often congenital, and, aside from the fatty growths found in spina bifida aperta and spina bifida occulta, are most frequently met with as large growths which are both intramedullary and extramedullary. In rare instances there are multiple growths which are both extradural and intradural, consisting of embryonic fat tissue (Fig. 98).

Dermoids, epidermoids, and cholesteatomas are unusual although a considerable number of cases have been reported in recent years. Gross (1928) reported one case and collected 19 from the literature. Two of the cases were extradural, 10 were intradural, and 6 were intramedullary. There were 4 extramedullary dermoids in my series of 275 spinal cord tumors. The growths were well encapsulated, contained caseous material and hair, and in 3 instances were adherent to the pial surface of the cord.

CHAPTER VIII

TUMORS OF THE SPINAL CORD, NERVE ROOTS, AND MEMBRANES

II. PATHOLOGY*

By Abner Wolf, M.D.

THIS STUDY IS BASED UPON THE GROSS AND HISTOLOGICAL EXAMINATION OF 345 SPECIMENS OF EXPANDING OR SPACE-TAKING lesions of the spinal canal and its contents. Of these, 253 are neoplasms; 81 are herniations of the nucleus pulposus, and/or thickening of the ligamentum flavum; 9 are inflammatory lesions; and 2 are abnormal changes in the vertebrae. The material is composed chiefly of tissue obtained at operation, although in some instances, autopsy preparations are also available.

Tumors which occur in and about the spinal canal are, in general, similar pathologically to those that are found in and about the cranial cavity. Like the intracranial lesions, they may arise from the neural parenchyma; from the membranes and bony structures enveloping the neural tissue (meninges, nerve sheaths, and vertebrae) ; from the vascular network in the osseous or connective tissue envelopes and in the spinal cord; from embryonal rests or inclusions in or about the spinal cord; or they may be metastases from tumors elsewhere in the body. In addition, there may be secondary deposits in the spinal canal of intracranial tumors, principally gliomas.

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* Some of the percentage frequencies given in this chapter and some of the views expressed are not in full agreement with statements made in the chapters devoted to the relative frequency, pathogenesis, symptoms, and treatment of tumors of the spinal cord and membranes. The differences may be due to the fact that the material upon which my figures are based included a number of cases from another hospital. The differences between the views expressed by Dr. Wolf and by me are small and few, but it has seemed advisable for each writer to express his personal viewpoint—C.A.E.

The present series was divided as follows:

Neoplasms of Spinal Cord and Canal

Primary glioma	26
Secondary glioma (implants from intracranial tumors)	11
Meningioma	57
Nerve sheath tumor (perineurial fibroblastoma, schwannoma, neurofibroma, neurinoma, neurilemmoma)	67
Hemangioblastoma	4
Chordoma	2
Dermoid cyst	2
Epidermoid cyst	1
Angioma	2
Vertebral lesions (primary):	
Angioma	2
Myeloma	4
Chondroma	3
Lipoma	4
Metastatic tumors:	
Carcinoma	28
Neuroblastoma (sympathicoblastoma)	1
Hypernephroma	2
Lymphoepithelioma	1
Sarcoma	32
Lymphogranuloma (Hodgkin's disease)	1
Chloroma (Myeloid chloroma)	1
Leukosarcomatosis of Sternberg (Lymphosarcoma + leukemia)	1

Other Space-Taking or Expanding Lesions Impinging on Spinal Canal

Herniation of nucleus pulposus and/or thickening of ligamentum flavum	81
Epidural tuberculoma associated with Pott's disease	5
Epidural abscess	3
Epidural actinomycosis associated with vertebral infection	1
Paget's disease	1
Exostosis of vertebra	1

Aside from the absence in the spinal canal of primary tumors arising from structures limited to the intracranial cavity, such as the choroid plexus, pituitary and pineal, the differences between intracranial and intraspinal neoplasms are chiefly a matter of relative frequency. The ependymoma, for instance, is one of the less common intracranial gliomas, while it is the most common intraspinal glioma. The nerve sheath tumor (perineurial fibroblastoma, schwannoma, neurinoma, neurofibroma, or neurilemmoma) is relatively more common than the meningioma in the spinal canal, although the reverse is true in the cranial cavity. Lipomas and chordomas, though rare in the spinal canal, occur there more frequently than they do intracranially.

In general, intracranial are more frequent than intraspinal tumors. For the present discussion the autopsy specimens for a twenty-year period, and the surgical preparations for eleven years have been reviewed. During these periods 4426 autopsies were performed (Presbyterian Hospital and Neurological Institute), and 2717 neurosurgical specimens (Neurological Institute) were examined. Only 253 primary and secondary intraspinal neoplasms were encountered, while 1569 intracranial neoplasms were seen.

Schlesinger (1898) found 150 spinal cord tumors in 3500 autopsies, which included both the primary and the metastatic lesions. The percentages were 0.43 per cent of all autopsies, and 2.06 per cent of all tumors. In the same material, there were 994 brain tumors. Antoni (1936) pointed out that tumors of the spinal cord and its surroundings were not frequent; but, in considering the ratio of 6 to 1 of brain tumors to spinal cord neoplasms, as found in Schlesinger's material, he indicated that this was not significant when one considers that the weight of the brain to that of the spinal cord is as 8 to 1. Peers (1936) in reporting on 10,592 autopsies (Boston City Hospital) found only 4 spinal cord tumors, all intramedullary, among 188 tumors of the central nervous system, or 2.1 per cent of the total.

Among the 253 spinal neoplasms considered here, the largest group is composed of nerve sheath tumors, although the metastatic tumors and the meningiomas approach them in frequency. Gliomas are only half as frequent and the other types of neoplasm occur only occasionally. The order is as follows:

	Number	*Per Cent*
Nerve sheath tumors (perineurial fibroblastoma, schwannoma, neurofibroma, neurinoma, neurilemmoma)	67	26
Metastatic tumors	64	25
Meningiomas	57	23+
Gliomas	26	10
Secondary gliomas	11	4+
Tumors arising from blood vessels	9	3+
Vertebral tumors (not including 2 angiomas or metastatic lesions)	7	3−
Congenital tumors (dermoid, epidermoid, chordoma)	5	2−
Lipomas	4	1.5
Malignant lymphogranuloma	1	0.5−
Chloroma (myeloid chloroma)	1	0.5−
Leukosarcomatosis of Sternberg (lymphosarcoma + leukemia)	1	0.5−

A similar tabulation of our intracranial neoplasms shows the following:

	Number	*Per Cent*
Gliomas	784	50
Meningiomas	242	15.4
Nerve sheath tumors	162	10.3
Metastatic tumors	162	10.3
Congenital tumors	83	5.3
Adenomas of pituitary	73	4.7
Vascular neoplasms	58	3.7
Skull and dural tumors	5	0.3

A consideration of other series as given in the literature reveals parallel findings. Cushing (1932) in reviewing the statistics in his series of 2023 intracranial tumors found the following order of frequency: Gliomas, 42.6 per cent; meningiomas, 13.4 per cent; nerve sheath tumors, 8.7 per cent.

Rasmussen, Kernohan and Adson (1939) classified 557 intraspinal neoplasms and listed: Neurofibromas (nerve sheath tumors), 163 or 29 per cent; meningiomas, 140 or 25 per cent; gliomas, 86 or 13 per cent.

NERVE SHEATH TUMORS

Nerve sheath tumors (perineurial fibroblastoma, schwannoma, neurinoma, neurofibroma, neurilemmoma, "peripheral glioma") are the most frequent of the neoplasms of the spinal canal, although followed closely in frequency by the meningiomas and the metastatic tumors. Sixty-seven of our 253 tumors of the spinal canal are of this type. It may be that the large number of nerve roots in the spinal canal, as compared with the more restricted total of cranial nerves, accounts for the difference in frequency between the spinal and intracranial sheath tumors, which is about 3 to 1. In any case, there is no predilection for the sheath of a single nerve in the spinal canal, as there is in the cranial cavity, where the eighth nerve is preponderantly involved. Any of the spinal nerve roots or nerves may be involved. Of the 67 nerve sheath tumors in our series, 25 were in the cervical spinal canal, 31 in the thoracic, 10 in the lumbar, and 1 in the sacral spinal canal. Some tumors extended over many vertebral levels, and in 2 of the 67 instances, there were multiple neoplasms so that this distribution is only an approximation. Rasmussen, Kernohan and Adson (1940) found that among their 167 similar tumors 30 occurred in the cervical spinal canal, 70 in the thoracic, 55 in the lumbar, and 2 in the sacral spinal canal.

Of our 67 nerve sheath tumors, 54 were intradural, 3 were both intra- and extradural, and 10 were extradural. Two of the combined intra- and extradural, and 6 of the extradural tumors protruded through eroded and enlarged intervertebral foramina. When there was a larger or smaller extravertebral mass connected with the intravertebral one by a pedicle the growth had the so-called dumbbell shape. This varying situation depends upon the fact that the sheath tumors may arise from the nerve roots intradurally, in their passage through the dura or from the nerves extradurally.

A single instance of an intramedullary nerve sheath tumor (listed as neurofibroma) occurred among the series of intraspinal neoplasms of Rasmussen, Kernohan and Adson. Histologically, this tumor resembled the other nerve sheath tumors and contained such a large number of connective tissue fibers that at one point in the discussion it was referred to as a "fibroma." This was not the "central neurinoma" of Josephy (1924) and MacPherson (1925), which had been demonstrated by Bailey and others to be a glioma (polar spongioblastoma), but a true sheath tumor.

These tumors may vary in size from small nodules up to masses which extend through six to eight vertebral levels and erode and enlarge the spinal canal. As already noted, some of the tumors may follow a nerve through an intervertebral foramen, eroding and enlarging it. When this occurs, it may force dura before it and not extend beyond the foramen; or it may become extradural and eventually extravertebral, extending into the neck, thorax, or abdomen (Fig. 111 A and B). The tumors may be globular, bulbous, oval, fusiform, or dumbbell-shaped. They are encapsulated and even-surfaced, nodular or irregularly lobulated. The capsule is firm, fibrous, and rough, or homogeneous and smooth; dull or glistening; grayish and relatively avascular, or grayish red or pink, brownish red, purplish, plum-colored and quite vascular. The firm capsule may at times be almost cartilaginous or, rarely, it may become soft and even friable. The central portion of the neoplasm varies from yellow to white with a red or green tinge; it may be firm, but it is usually soft, and often friable and cystic. There may be many small cysts or one large one, containing clear yellowish fluid or gelatinous or glairy material. The yellow central portion of the tumor may shine through the thin capsule and give the surface of the tumor a yellowish tint.

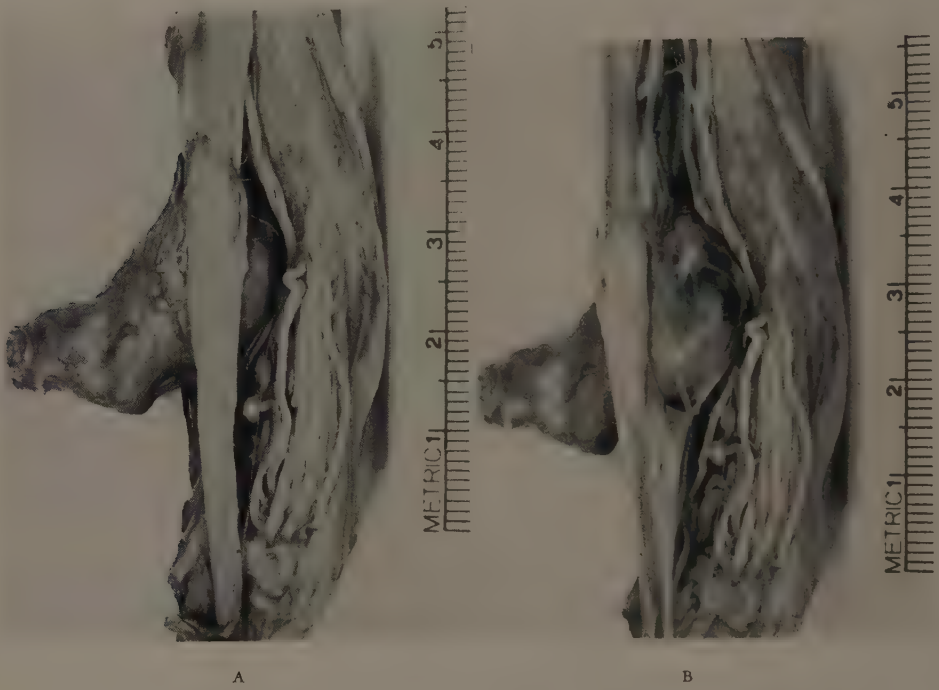


FIG. 111. Nerve sheath tumor (perineurial fibroblastoma). The tumor was attached to the nerve roots of T 12; it was both intradural and extradural and passed out through the dura along T 12. Note the compression of the segments of the spinal cord.

Histologically, the tumor capsule is composed of compact fibrous tissue which, relatively, is sparsely cellular. The cells have the ap-

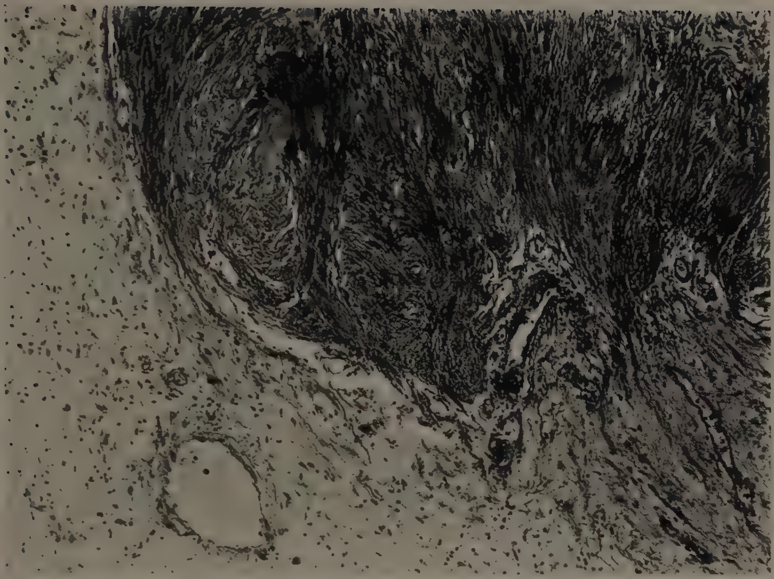


FIG. 112. Nerve sheath tumor (perineurial fibroblastoma). The neoplasm was intradural and lay within the spinal canal from T 9 to L 1, being right ventral in position. The tumor was cystic grossly. Note the characteristic solid and loose-meshed zones in the tumor and the interlacing bundles of long slender cells in the solid area. The loose-meshed area is sparsely cellular. Hematoxylin eosin stain. ×110.

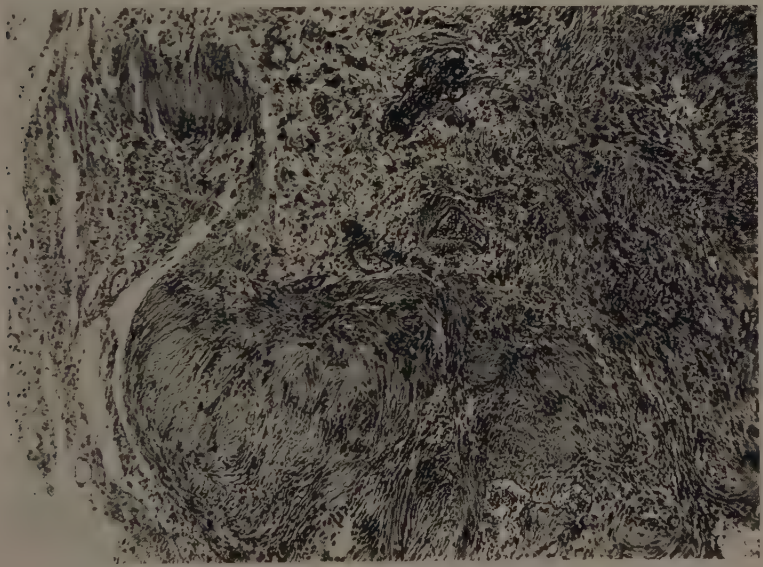


FIG. 113. Nerve sheath tumor (perineurial fibroblastoma) in the spinal canal attached to the right ventral nerve root of C 3, which lay both intradural and extradural and extended from C 2 to C 4. The tumor contains characteristic compact and loose-meshed areas. In the solid zone note the palisades and some lunate-layered formations each producing somewhat the effect of a transected onion bulb. Hematoxylin eosin stain. ×110.

pearance of ordinary adult connective tissue cells, and the fibers are a rich collection of collagen and reticulin. The vascularity of the capsule varies; at times it may be highly vascular. The body of the tumor consists of compact and loose-meshed tissue (Fig. 112). In 45 of the 67 neoplasms in the present series, the solid areas constitute two-thirds or more of the tissue examined, in 13 the loose-meshed zones predominate to a similar degree, while in 8 there are equal amounts of each type of tissue. The two types may be evenly interspersed so that the solid areas appear as islands in the loose tissue (Fig. 113), or one sort of tissue may predominate.

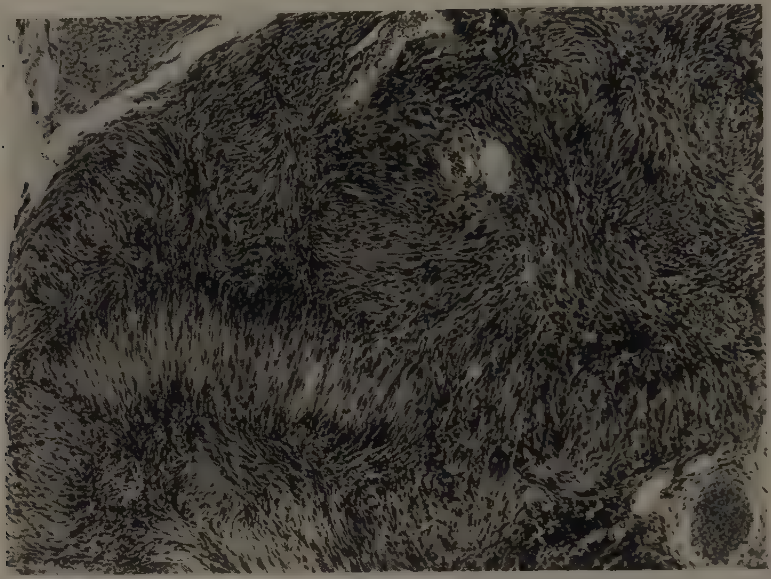


FIG. 114. Nerve sheath tumor (perineurial fibroblastoma). The neoplasm was intradural, extramedullary and attached to the right dorsal nerve root of T 4. The highly cellular tumor contains long slender cells which are closely approximated and arranged in parallel rows in columns which curve and interweave or in short typical palisades. Nuclei of neoplastic cells are hyperchromatic and elongated, showing an apparent variation in size and in shape to some extent. Hematoxylin eosin stain. ×140.

The compact areas (Type A, or fibrillar tissue of Antoni, 1920) are highly cellular and have a characteristic architecture. The elongated cells are arranged in parallel rows in columns which have a tendency to curve. These columns may stream in any direction and interweave and intertwine, so that in a single microscopic field, the pattern may be complex, due to the various angles at which columns are cut (Fig. 114). Single cell columns, especially in the smaller islands of solid tissue, may be relatively short but quite broad and somewhat bowed, producing an "accordion effect" (Fig. 114). They may also be arranged in globular or ovoid masses in which parallel rows of cells may bow away from the center of the mass in concentric layers, producing an "onion bulb" effect (Fig. 113). The tumor cells are elongated and narrow, and their cytoplasm is finely granular or homogeneous, and lightly acidophilic. The nuclei are also quite long, oval, and slender. They have a prominent nuclear membrane and are hyperchromatic, containing a good deal of finely granular chromatin and occasionally nucleoli (Fig. 115). At times, clusters of nuclei may be lighter-staining. Mitoses are infrequent.

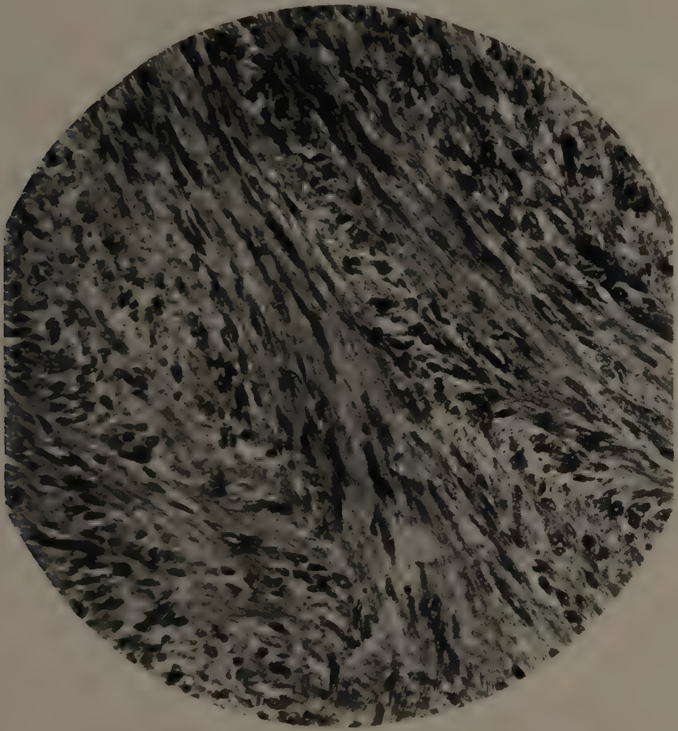


FIG. 115. Nerve sheath tumor (perineurial fibroblastoma). The neoplasm was intradural and attached to the right dorsal nerve root of T 9. It contains columns of elongated cells arranged in parallel rows. Notice the abundance of connective tissue fibers in these columns, the slender nuclei, and the variability of nuclear shape and size. Phosphotungstic acid-hematoxylin stain. ×600.

The shape and size of the nucleus may appear to be variable due to the curving and eddying of the columns of parallel cells so that the nuclei are cut at various angles (Fig. 116).

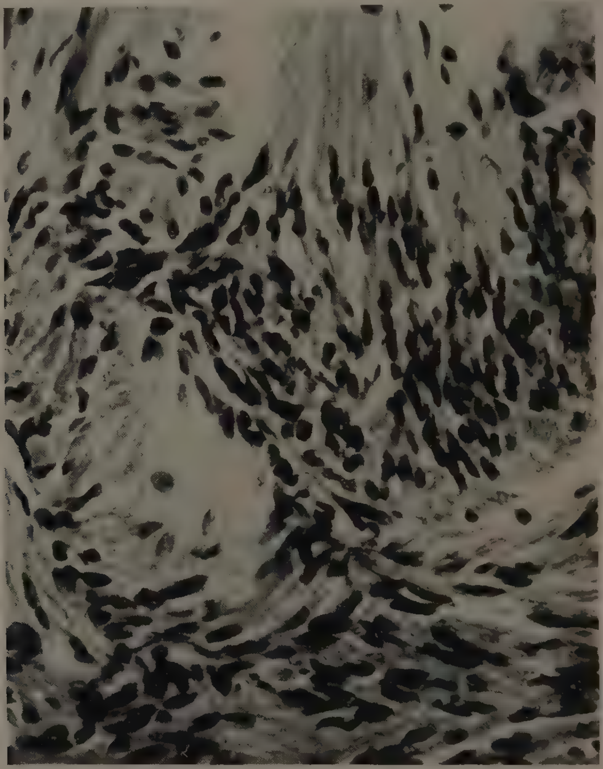


FIG. 116. Nerve sheath tumor (perineurial fibroblastoma). The neoplasm was both intradural and extradural, extended from C 2 to C 4 and was attached to the right ventral nerve root of C 3. The nuclei of the tumor cells are hyperchromatic and show apparent variability in size and shape although the majority are obviously long and slender. Hematoxylin eosin stain. ×460.

A most striking feature, and one which is of great aid in identifying this neoplasm, is the tendency of these slender, oval nuclei to cluster and to be arranged in rows strung out at right angles to their long axes in a single transverse plane "like staves of a barrel" (Verocay). This architectural feature has been termed a palisade (Figs. 113, 114, 117), and, following Francini, Verocay and Antoni, it has been accepted as pathognomonic of nerve sheath tumors. The cytoplasm streams away in similar parallel fashion from about either end of the nucleus amidst sheaves of fine reticulin fibers and frequently a second row of nuclei forms a second parallel palisade adjacent to the first (Fig. 117). In some of these sheath tumors, such palisading, occurring very frequently at regular intervals and roughly in parallel long curving columns of cells, produces a "step-ladder" effect (Fig. 118). More than two-thirds of the 67 tumors have palisades and in half of these they are present in great profusion. The remaining group of less than one-third show no definite palisading, although there is often suggestive nuclear clustering.

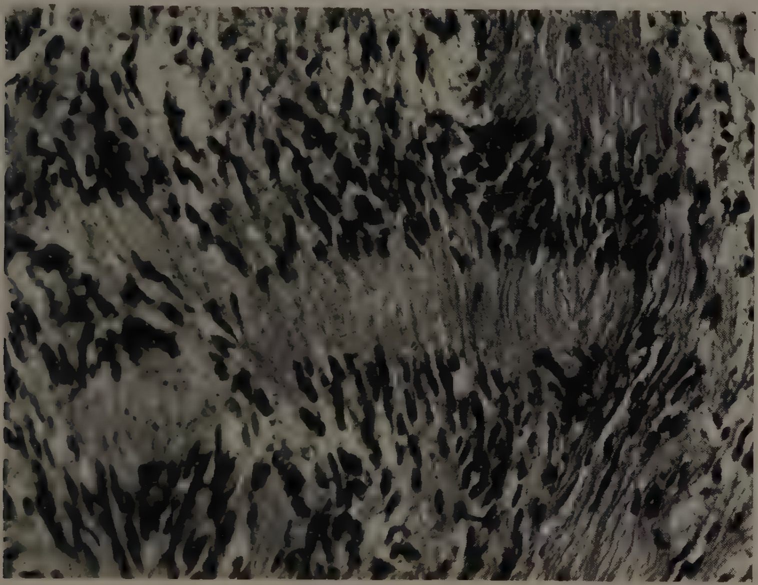


FIG. 117. Nerve sheath tumor (perineurial fibroblastoma). The neoplasm was intradural and extradural, extended from C 2 to C 4, and was attached to the right ventral nerve root of C 3. The long slender cells with long cell processes are arranged in parallel rows. There is a tendency for the nuclei to cluster in rows, producing palisades. Many fine connective tissue fibers are present between the cells, running parallel to their long axes. Hematoxylin-eosin stain. ×460.

Another prominent and characteristic feature of this type of tumor is the profusion of connective tissue fibers in the solid areas. These are fine collagen and reticulin fibers which lie between the individual elongated tumor cells and run parallel to their long axes (Fig. 119). As seen in a reticulin stain, these very regularly arranged, closely approximated, parallel fibers produce a compactly fibrillar picture which faithfully reproduces the architecture of the curving and interweaving columns of tumor cells and is unmistakable (Fig. 120). In relation to the palisade formations, the number of fibers tends to be sharply diminished or perhaps diverted about

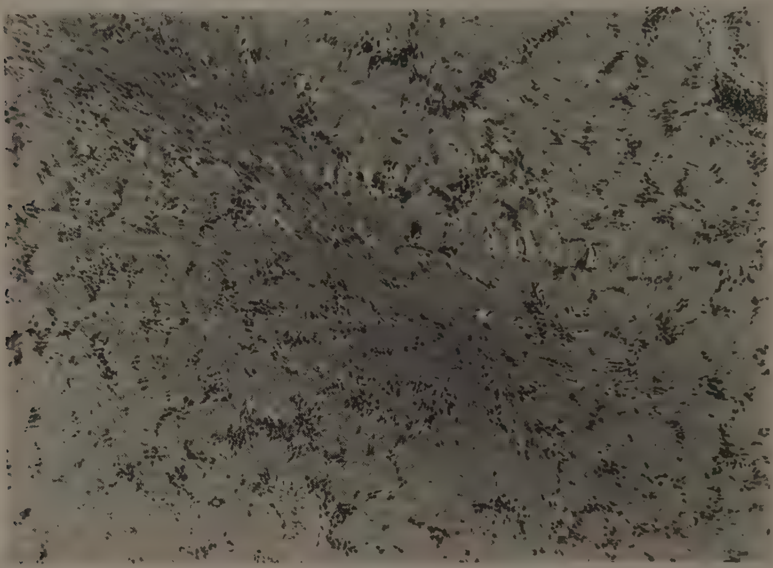


FIG. 118. Nerve sheath tumor (perineurial fibroblastoma). The neoplasm extended from C 6 to T 1 and was attached to the ventral root of C 7. This was a "dumbbell" shaped tumor which was intradural and extradural, and extravertebral, and extended into the neck through the intervertebral foramina of C 6, C 7 and T 1 on the right. The tumor tissue is richly fibrous and the cell outlines are not clear. The nuclei are seen to cluster in a definite stepladder pattern. The individual rows of nuclei mark the palisades. Hematoxylin eosin stain. ×140.

the cells at the level of the nuclear clustering so that transverse spaces in the fiber streams give the negative picture of the palisade (Fig. 121).

The loose-meshed tissue (Type B, or reticulated tissue of Antoni, 1920) is sparsely cellular in contrast to the richly cellular compact areas. It has a rather myxomatous appearance. The cells which compose it are elongated, slender cells of the type described in the compact zones, or they may be irregular or roughly stellate (the star-shaped cells may resemble astrocytes but show none of the specific staining reactions of such glial cells). Their nuclei are oval and either long and slender or short and hyperchromatic. The cells are often shrunken and malformed, and their nuclei pyknotic, having evidently undergone degeneration. Between the cells, amorphous-homogeneous or finely granular material is present. There may be small or moderate numbers of reticulin or collagen fibers spread well apart (Fig. 120) but often there is none. Swelling, curling, and fragmentation of such fibers are not infrequent. Clusters of large, oval, or rounded cells with translucent, finely or coarsely vacuolated cytoplasm and small spherical or oval, hyperchromatic, central or eccentric nuclei may be encountered (Fig. 122). The nuclei may have an irregular outline due to indentation by globules of lipoid which fill the vacuolar spaces and include neutral fat. Slow degeneration of the tissue is evidently in progress and these lipoid-laden mononuclear cells are associated with that process. They vary in frequency and numbers and are not usually a prominent or even constant feature in sections of fixed tissue. (They are seen with great regularity in

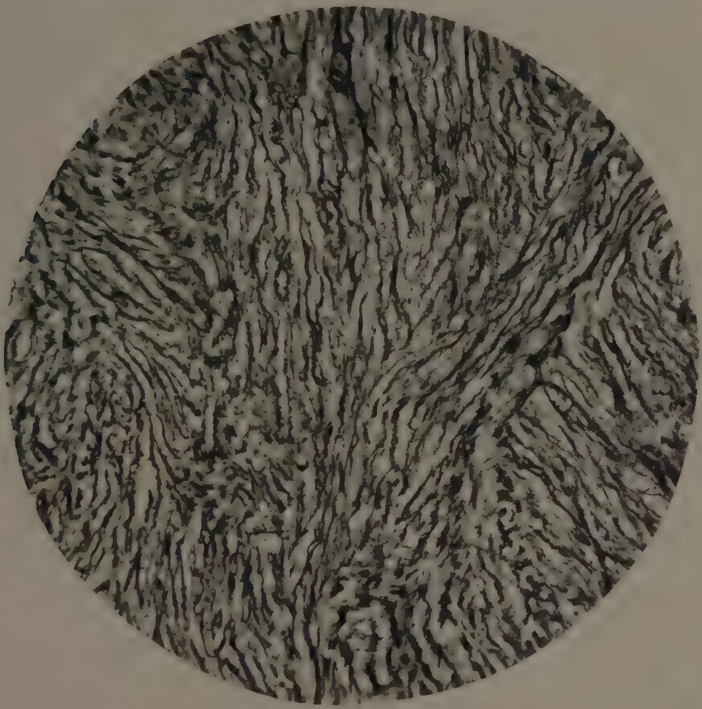


FIG. 119. Nerve sheath tumor (perineurial fibroblastoma). The neoplasm was intradural, attached to the right dorsal nerve root of T 9. An abundance of connective tissue fibers (reticulin) may be seen in the compact zone of the tumor. Note the regular arrangement of fibers in bundles corresponding to the interweaving cell columns. Laidlaw connective tissue stain. ×460.

tumors of this type in fresh tissue or supravital preparations, while, curiously, they are detected with difficulty ordinarily in fixed preparations.) The number of such cells is probably dependent upon the stage of the degenerative process reached at the time the tumor is examined.

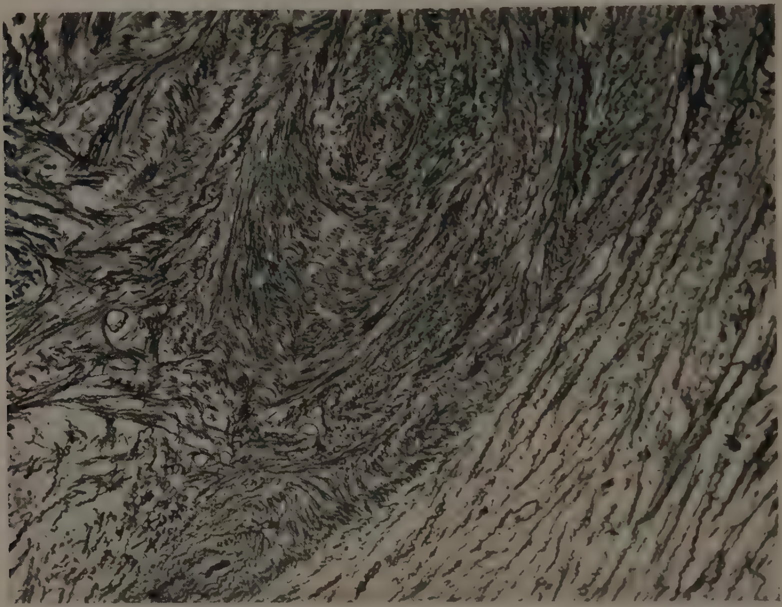


FIG. 120. Nerve sheath tumor (perineurial fibroblastoma). The neoplasm was intradural and extradural, extended from C 2 to C 4, and was attached to the right ventral nerve root of C 3. There is a rich reticulin network in the compact area and relatively few reticulin fibers in the loose-meshed zone in which the cells are also fewer. Laidlaw connective tissue stain. ×110.

Zones of transition from compact to loose-meshed tissue are easily found and it seems clear from their appearance and from that of edematous foci in long stretches of compact tissue, that the loose-meshed areas are simply a stage of degeneration of the compact zones. The edema spreading cells and fibers apart, the degeneration of these elements rendering the tissue sparsely cellular and fibrillar and distorting those that remain, and the products of degeneration in these transitional areas, are the histological features in support of this view. At times, hemosiderin-laden phagocytes and perivascular lymphocytes are present in addition to the lipoid-laden cells.

The occurrence of cysts in the solid areas in many of these tumors is another feature of this degenerative process. Fifteen of the 67 sheath tumors under consideration contain varying numbers of cysts. Many of these are microscopic, while others are large. The cysts are lined by tumor cells which may have their long axes parallel, or at right angles, to the margin of the cyst cavity (or running parallel to this margin but toward the eye producing a pseudo-epithelial appearance) (Fig. 123). There are usually three to four solid rows of cells and loose-meshed tissue beyond. Cells may be at any angle to the wall but usually a group or groups of elements are oriented the same way. Within the lumina of the cysts, there are often amorphous acidophilic material, degenerated tumor cells, and large mononuclear phagocytes.

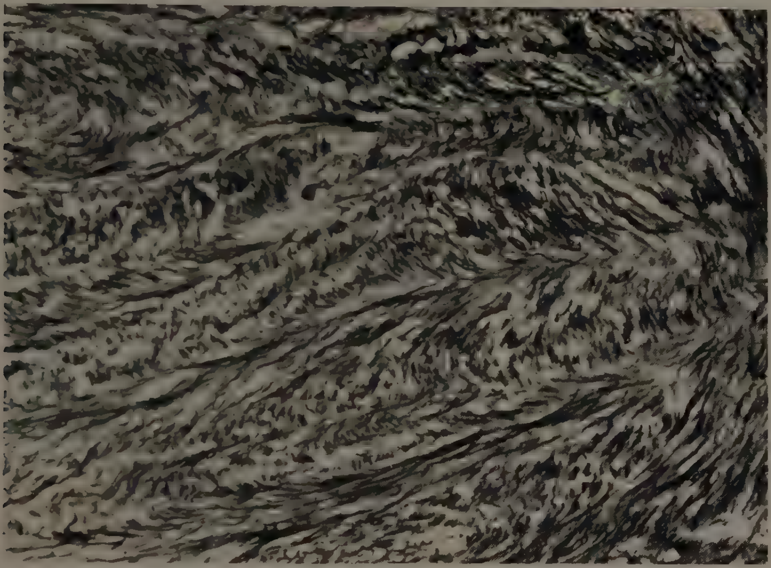


FIG. 121. Nerve sheath tumor (perineurial fibroblastoma). The neoplasm was intradural and extradural, extended from C 2 to C 4 and was attached to the right ventral nerve root of C 3. There is a rich connective tissue meshwork. Fine reticulin fibers occur between the parallel rows of tumor cells and reproduce the pattern of the interweaving columns of neoplastic elements. Laidlaw connective tissue stain. ×300.

Rasmussen, Kernohan and Adson (1940) in a review of 163 intraspinal nerve sheath tumors ("neurofibromas") state that most of these tumors within the spinal canal have a tendency to degenerate and become cystic leaving at times only a nubbin of tumor tissue. The cysts are usually filled with yellow fluid and when markedly distended become firmly adherent to the dura.

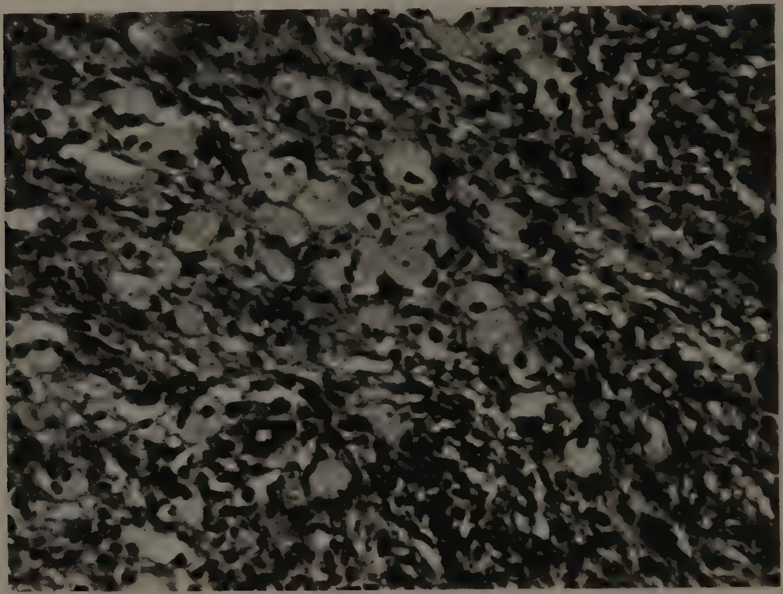


FIG. 122. Nerve sheath tumor (perineurial fibroblastoma). The neoplasm was intradural and extended from C 2 to C 5. There is a cluster of large, light-staining, lipoid-laden cells in the compact zone. They have eccentric, hyperchromatic, spherical, oval or irregular nuclei. Hematoxylin eosin stain. ×230.

About three-quarters of the 67 sheath tumors are moderately or richly vascular, the vessels being predominantly capillaries. In addition to small numbers of arterial and venous channels, some thin-walled sinuses are present. The remaining one-fourth of these tumors is relatively poor in vessels, although even in these, clusters of capillaries may be found. Within the loose-meshed zones and in relation to the cysts, the blood vessels often show abnormalities of their walls. There is swelling and multiplication of the endothelial cells of the capillaries with an increase of the subendothelial fibrous tissue (Fig. 124). The precapillaries and arterioles may show similar endothelial changes, fibrosis, and thickening, and at times partial hyalinization of their walls with partial or complete occlusion of their lumina (Fig. 125). Occasionally endothelial hyperplasia (and subendothelial lipoid-laden cells) are seen in a small artery, and at times there is mural and perivascular infiltration by lymphocytes, large mononuclear cells and, rarely, eosinophiles. It may be that these abnormal vessel changes account for the formation of cysts and the loose-meshed tissue, or they may be an associated phenomenon.

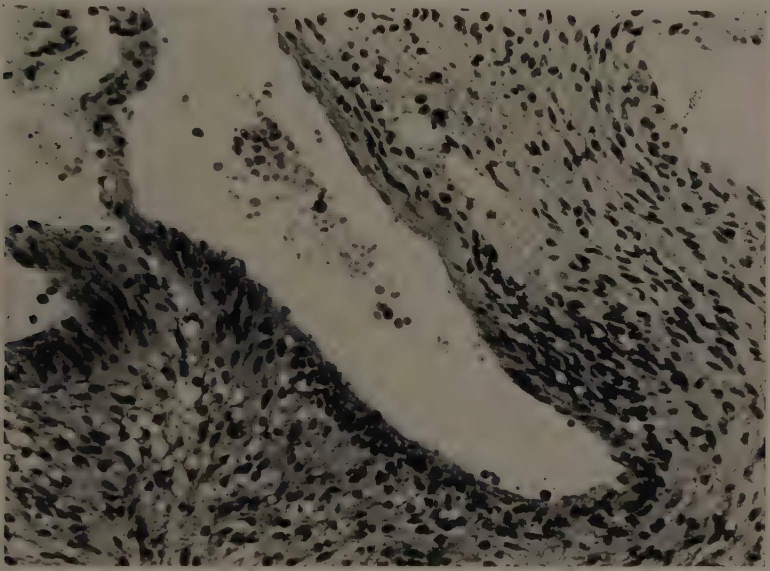


FIG. 123. Nerve sheath tumor (perineurial fibroblastoma). The neoplasm was extradural, extended from T 6 to T 7, "dumbbell" in shape, and extended into the thorax through the right intervertebral foramen between T 6 and T 7. Cystic areas are present in the tumor and tumor cells form the walls of the cysts. When the long axes of those cells run at right angles to the surface of the cyst walls a pseudo-epithelial appearance, as seen in one area, may be produced. Hematoxylin eosin stain. ×300.

As has been said, the connective tissue fiber content of these tumors, particularly their solid areas, is very high. In some instances, but not commonly, the degenerative process described is accompanied or followed by an even more striking accumulation of such fibers. There may be intense fibrosis in islands about the abnormal vessels; or large stretches of neoplasm, usually in the wall of a large cyst, may show a marked reduction in the number of tumor cells with dense fibrous tissue predominating and compressing and constricting islands of persisting neoplastic elements. This tissue may become partially hyalinized and may appear somewhat myxomatous.

There is a group of nerve sheath tumors, usually referred to as *neurofibromas* [although Rasmussen, Kernohan and Adson (1940) use this term for all the nerve sheath tumors, and Antoni (1936), Bailey and Herrmann (1937) and Tarlov (1940) tend to treat them as one], which differ from the others in that nerve fibers are found passing through them. Only one instance of such a tumor occurs among our 67, and, as usual, this is from a case of neurofibromatosis or von Recklinghausen's disease. The nerve fibers are continuous with those in the spinal nerve root or nerve involved, and may be myelinated or unmyelinated (Fig. 126). The other features may be very much like those in the rest of the nerve sheath tumors, although loose-meshed areas usually predominate and columns of cells are long, slender, and ordinarily spread well apart (Fig. 127). Palisading may be present, although it is not common, and the connective tissue fiber content is high, although its pattern is not as regular, due to the predominance of loose-meshed zones. Transitions may occur

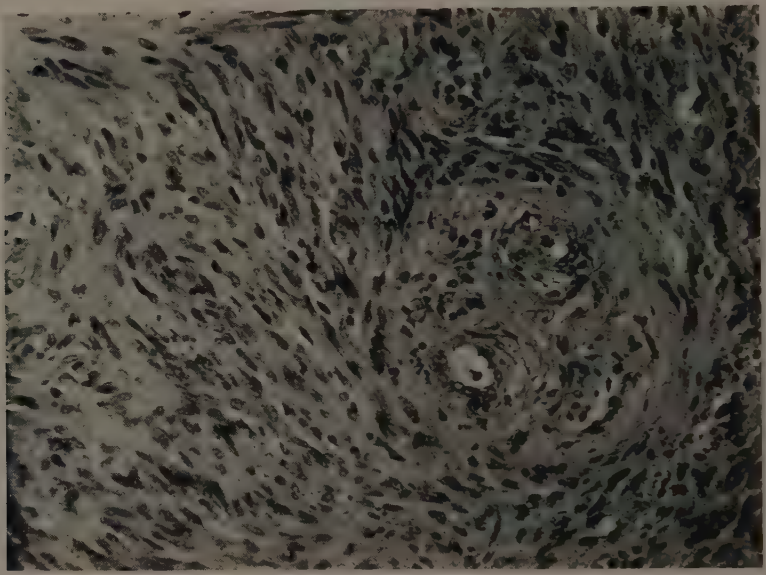


FIG. 124. Nerve sheath tumor (perineurial fibroblastoma). The neoplasm extended from T 12 to L 5 and was both intradural and extradural. Note the endothelial hyperplasia in a capillary and an arteriole in a compact area. Hematoxylin eosin stain. ×300.

between these "neurofibromas" and the rest of the group of sheath tumors, although many prefer to retain them in a separate category, since they are usually multiple and are often associated with other tumors and congenital malformations in and outside of the central nervous system.

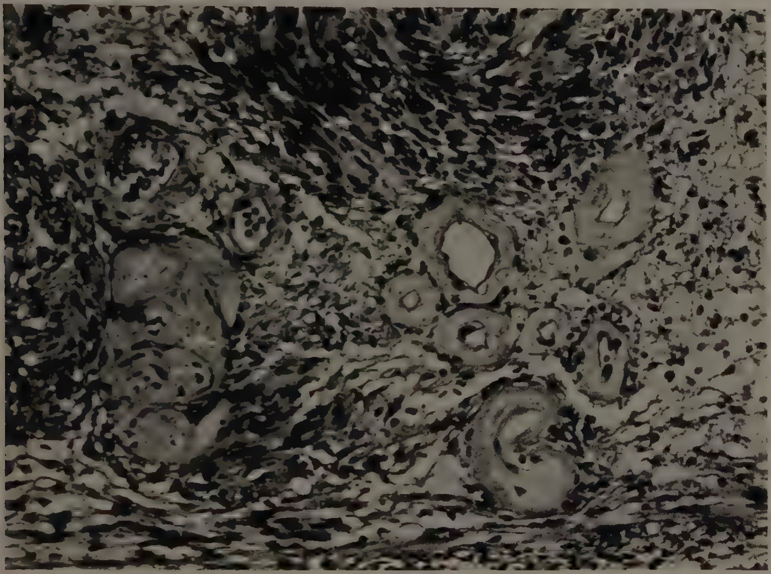


FIG. 125. Nerve sheath tumor (perineurial fibroblastoma). The "dumbbell" shaped neoplasm was extradural, between T 6 and T 7 and extended into the thorax through the right intervertebral foramen between T 6 and T 7. Note the fibrosis, thickening, and partial hyalinization of the walls of capillaries and arterioles in a loose-meshed area resulting in a reduction in size of their lumina. The loose-meshed tissue is edematous and degenerated. Hematoxylin eosin stain. ×300.

The origin of this whole group of nerve sheath tumors is still in dispute. All agree that these neoplasms arise from the nerve or nerve root sheath, but there is still disagreement as to whether they originate from neuroectodermal cells, the Schwann cells, or from the mesodermal elements of the perineurium. Bard, Francini, Verocay, Antoni, Masson, and others support the Schwann cell theory. Verocay (1908) used the term neurinoma to indicate the neural origin of the tumor. Antoni (1920) used lemmoma or neurilemmoma to indicate its origin from the specific neuroectodermal sheath, the neurilemma or Schwann sheath, postulating a precursor cell, the "lemmoblast" in the neural crest. Masson (1932), following a number of the investigators mentioned, referred to these tumors as Schwannomas (the "peripheral gliomas" of Roussy, Lhermitte and Cornil, 1924) for the same reason. Masson believed that palisading in these tumors was a manifestation of typical multiplication by longitudinal splitting of Schwann cells with clustering of the nuclei. He further believed that the derivation from Schwann cells could be followed in experimental neuromas ("experimental Schwannomas") and that the Schwannian syncytium was clearly reproduced in nerve sheath tumors. A most interesting recent contribution to the subject was the tissue culture study of Murray and Stout (1940). They cultivated cells from normal human and animal, fetal and adult nerves, from experimental neuromas ("experimental Schwannomas"), and from two instances of peripheral nerve sheath tumor. From the tumors, they grew out slender cells, resembling elements grown from the other sources, which were considered to be Schwann cells. No fibroblasts grew from the tumors, although they had grown abundantly from the other sources. These investigators concluded, therefore, that the primary tumor cell was a Schwann cell. Another most interesting conclusion was that there were two types of Schwann cells in their tissue cultures. These they designated A and B cells, corresponding to Antoni's Type A and B areas of a neurinoma (nerve sheath tumor) or to the compact and loose-meshed

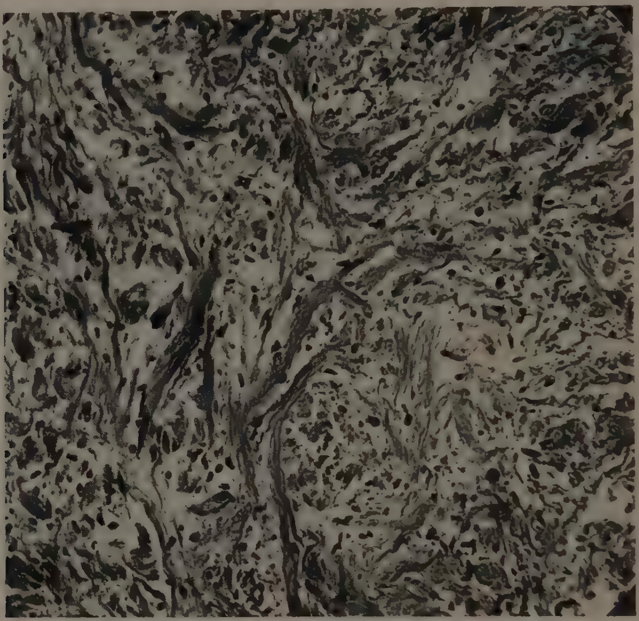


FIG. 126. Nerve sheath tumor (neurofibroma). The neoplasm was intradural and extradural, extended from C 5 to C 6 on the left and was attached to a nerve root of C 6. Note the separated strands of nerve separated by tumor cells. Eosin methylene blue stain. ×300.

areas referred to in our description. The B type of cell in tissue culture showed a marked tendency to digest the fibrin clot in which it had grown or into which it had migrated. It was suggested that the degenerated, microcystic appearance of the loose-meshed or Type B areas in nerve sheath tumors might be due to abnormal hyperactivity of this digestive, liquefactive function of the Type B cell acting on collagen and reticulin fibers and on other cells. That it was not merely a primary degeneration of the cells, as suggested by Antoni and by this author, was thought to be indicated by the vigorous, apparently unimpaired growth of B type cells in tissue culture.

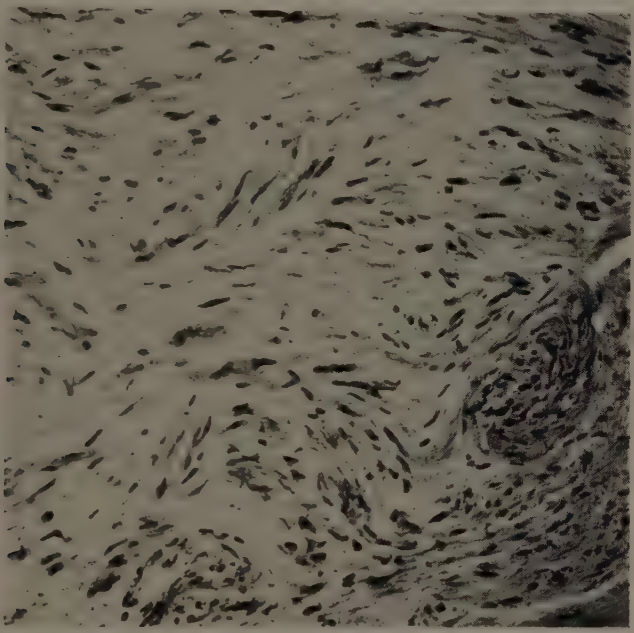


FIG. 127. Nerve sheath tumor (neurofibroma). The tumor was extradural, extended from C 2 to C 6 and was attached to the ventral roots of C 3 and C 4. Note the small nerve bundle at the left embedded in the heart of the neoplasm. The tumor cells are elongated and have long hyperchromatic nuclei. The tumor tissue is loose-meshed and myxomatous. Eosin methylene blue stain. ×300.

Von Recklinghausen (1882) suggested the connective tissue origin of nerve sheath tumors. Cushing (1917), in considering the intracranial nerve sheath tumors, specifically the eighth nerve or cerebellopontine neoplasms, referred to them as acoustic neuromata or neurinomata, and stated that they probably represented a transition between neuroglia and connective tissue. Mallory (1920 and 1929) proposed that the tumor be called a "perineurial fibroblastoma," implying that it arises from the mesodermal or connective tissue sheath about the nerve, and that the primary cell is a fibroblast. The wealth of connective tissue fibers present in these tumors is considered to be the point of proof and depends upon the theory that collagen and reticulin is formed exclusively by cells of mesodermal origin. Masson (1932) and others, and lately Murray and Stout (1940), however, feel strongly that such fibers can be laid down under the influence of Schwann cells, neuroectodermal elements. Penfield (1917) supported Mallory's view, and recently Tarlov (1940), using a modification of Hortega's silver stain, presumably specific for Schwann cells, reports that he is unable to demonstrate Schwann cells in nerve sheath tumors and concludes that they are of perineurial or endoneurial origin. Bailey and Herrmann (1937), after a careful study of the tumors in two cases of von Recklinghausen's disease, remain in doubt as to the two theories, and choose the noncommittal term, neurilemmoma, as proposed by Stout, to indicate a tumor of the nerve sheath without implying a neuroectodermal origin. The use of the noncommittal term, nerve sheath tumor, here implies the same doubt. The origin of the spinal intramedullary nerve sheath tumors of Rasmussen, Kernohan and Adson (1940), and others cited, is a puzzle. Depending on which theory of origin of nerve sheath tumors one favors, one may derive them from misplaced mesenchyme destined to become endo- or perineurium, or from misplaced neural crest tissue.

MENINGIOMA

Meningioma (dural sarcoma, psammoma, dural endothelioma, arachnoidal fibroblastoma, meningeal fibroblastoma, meningothelioma, meningoendothelioma, pachymeningioma, leptomeningioma). Of the 253 neoplasms of the spinal canal in this series, 57 are meningiomas. As has been pointed out, they are somewhat less frequent in the spinal canal than are the nerve sheath tumors, the ratio in this group being 57 to 67 or 23 per cent as compared to 26 per cent of the total.

This relative frequency is similar to that found by Rasmussen, Kernohan and Adson (1940) who, among 557 intraspinal neoplasms, found 140 meningiomas and 163 nerve sheath tumors, or 25 per cent as compared to 29 per cent. Learmonth (1927), on the other hand, found the meningiomas somewhat more frequent (55 per cent of juxtamedullary spinal neoplasms), and Antoni (1936) believed them to be at least as frequent.

More than half of the 57 meningiomas in this series extended over two or more vertebral levels of the spinal canal, 7 extending over four vertebral levels. Sixteen of these neoplasms occurred in the cervical spinal canal, 39 were thoracic, one was lumbar, and one was lumbosacral. Four of the high cervical tumors passed through the foramen magnum. Fifty-three tumors were intradural, 2 were both intra- and extradural, and 2 were wholly extradural. No "dumbbell" tumors were encountered in this group.

Of the 140 spinal meningiomas of Rasmussen, Kernohan and Adson, 23 or 16.5 per cent were in the cervical spinal canal, 115 or 82 per cent were thoracic, and 2 or 1.5 per cent were lumbar. In this same series, 130 or 93 per cent were intradural and 10 or 7 per cent intra- and extradural. Among Learmonth's 67 spinal meningiomas 13.3 per cent were cervical, 63.4 per cent thoracic, and 23.3 per cent were lumbosacral.

Thirty-seven of our own 57 spinal meningiomas were dorsolateral in position, 18 were ventrolateral, and 2 almost completely surrounded the spinal cord. This is in partial contrast to the nerve sheath tumors in which the dorsolateral position is even more frequent. The great majority of the 57 spinal meningiomas are described as being firmly attached to the dura, at times over a broad surface, and often secondarily attached to the dentate ligament, to the nerve roots coursing over them, and to the arachnoid membrane. Instances in which no dural attachment is present have been reported (Herzog 1909; Elsberg et al., 1931). These tumors without dural attachment correspond to the similar intracranial neoplasms, occurring in the choroid plexuses, deep in the Sylvian fissures, and in a juxtacerebellar position (Cushing and Eisenhardt, 1938). In 3 of the present series of 57 spinal meningiomas, the dura was found to be invaded histologically. Since this material is wholly surgical, this figure is not representative, and moderate invasion of the pachymeninx must be as frequent as it is in the cranial cavity, as an observation on the gross appearance of the tumor (page 254) will show. Antoni (1936) stated that the spinal meningioma almost always grows diffusely between the dural layers. An extradural tumor of our series invaded a vertebra, and one intradural tumor which invaded dura extensively also invaded the overlying vertebra. Both extradural neoplasms invaded the epidural fat.

The spinal meningioma may be rounded, flattened, irregular, lobulated or nodular, and its surface may be smooth, granular or fibrous. The tumor varies in size from a small nodule to a large mass extending over many vertebral levels or extending as a large tumor into the posterior fossa.

The capsule, which is tough and fibrous, and some of the connective tissue trabeculae which arise from it and traverse the neoplasm, seem to be of dural origin. Thin spicules of calcium or bone may be present in the capsule, and rarely the capsule may be almost cartilaginous or soft and friable. It is dull or glistening, reddish or bluish gray, and usually quite vascular.

The body of the tumor is dense and usually soft, but occasionally it is firm. Its cut surface is granular, fleshy or fibrous. Fine gritty particles are frequent in it and coarser spicules of calcium, bone (in 2 of our 57 cases), and rarely cartilage may also be present. The body of the tumor may be plum-colored or may vary from red to gray, and it is relatively avascular.

Histologically, these tumors form a distinct and uniform group, the divergent appearances of which are less striking than their resemblances. They will, therefore, be described as a unit, outlining the variations within the group and their frequency. There has been a renewed tendency in recent years to divide this group (Bailey and Bucy, 1931; Globus, 1935; Cushing and Eisenhardt, 1938) into subgroups, depending upon resemblances to embryological stages of the development of the leptomeninges; the tendency to occurrence or predominance of specialized connective tissue structures in these tumors, such as reticulin, collagen, cartilage, bone, and so on; the predominance of blood vessels; evidence of origin from one unit of the meninges (as the pia in melanoblastomas); and the like. In this type of classification, all tumors which arise from the pachymeninges or leptomeninges are included under the designation meningioma, as well as some tumors, such as extradural and intraparenchymal lipomas, which may not be of meningeal origin.

It is proposed here to limit the term meningioma to that large and characteristic group of tumors which, it is now fairly generally agreed, arise from arachnoidal granulations. (The origin of these tumors is discussed on page 263.) When originally proposed by Cushing, the term meningioma was used in this narrow sense. It would seem wise to designate the other rarer neoplasms of the meninges as meningeal melanoblastoma, meningeal sarcoma, and the like, depending upon their special characteristics.

FIG. 128

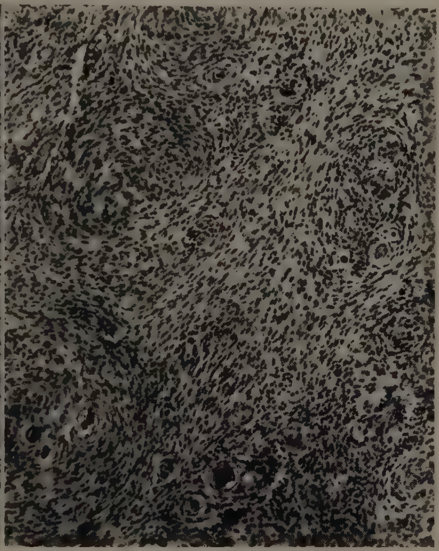


FIG. 129

FIG. 128. Meningioma. The neoplasm was intradural and extended from T 2 to T 3 on the left. The tumor tissue is highly cellular and its cells are arranged in parallel rows in columns which curve and are of varying length. They are chiefly elongated elements with oval, uniform nuclei. The cell outlines are obscured in places, producing somewhat the appearance of a syncytium. Hematoxylin eosin stain. ×110.

FIG. 129. Meningioma. The neoplasm was intradural at T 9. Parallel rows of elongated cells form indefinite columns which often curve. Short single rows of tumor cells in circles are arranged in concentric layers resulting in whorl formation. Some of these whorls are partially calcified. Hematoxylin eosin stain. ×110.

Of the 57 spinal meningiomas of the present series, 42 are very highly cellular, 13 moderately so, and 3 sparsely cellular. The architecture of these tumors varies, but only to a minor degree and no more than it frequently does within a single neoplasm. In general, meningiomas impress one by their uniformity. In 44 of the tumors, elongated cells are found arranged in parallel rows, in short or long columns which curve (Fig. 128), but which, unlike the somewhat similar structures in nerve sheath tumors, do not usually intertwine or interweave. The columns are closely approximated and may give the appearance of a continuous sheet of cells (Fig. 129). The individual neoplastic elements are elongated, bipolar, or fusiform cells with moderate or fairly abundant fine granular, acidophilic cytoplasm and indistinct or fairly definite outlines (Fig. 134). They are stouter, in general, than the cell of the nerve sheath tumor. Their nuclei are oval, uniform, central, and characteristically light-staining (Fig. 134). They have a heavy nuclear membrane and only a small amount of finely granular chromatin spread throughout the rather translucent nucleus. They differ strikingly from the long, slender, hyperchromatic nucleus of the nerve sheath tumor with its apparent variability in size and shape (Fig. 116). Mitoses are absent. These columns of cells, though present in over three-fourths of our meningiomas, are the predominant structure in only 6; they are equal in frequency to other characteristic architectural features described below in 14 instances; while in the rest they are relatively inconspicuous. In 19 of the 57 neoplasms, islands of cells (Fig. 130)

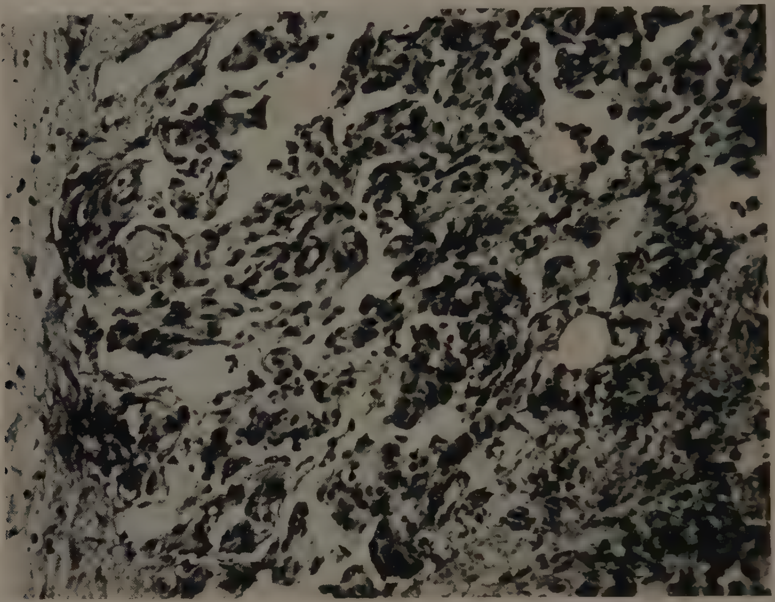


FIG. 130. Meningioma. The neoplasm was extradural and extended from C 7 to T 2. The tumor shows a tendency toward the formation of cell islands interconnected by cellular bridges reproducing the structure of arachnoid villi. Hematoxylin eosin stain. ×230.

occur which are partially or completely separated from each other by the short curving columns of cells or in some instances by fine, vascular, connective tissue trabeculae. The cells are essentially like those described above in the columns, except that they are often stouter, more polygonal. These islands of cells have a strong resemblance to the globular, oval, fusiform, or lunate arachnoidal granulations, and are frequently connected to one another and to the cell columns by cellular bridges (Fig. 130). The close approximation of these structures at times obscures their outlines. In 3 tumors, large uniform sheets of cells are seen, not showing the parallelism of the columns or the clustering in small groups of the cell islands. The individual elements are predominantly polygonal and may be closely approximated and poorly outlined, or slightly separated and sharply limned, but in every other essential they are like the tumor cells previously described. In addition, these 3 neoplasms present all the other architectural arrangements so far described.

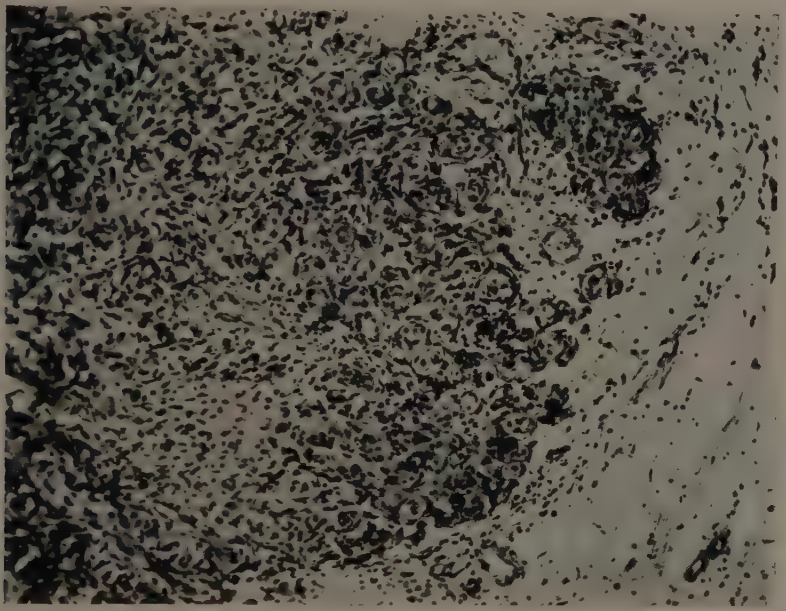


FIG. 131. Meningioma. The neoplasm was extradural and extended from C 7 to T 2. Small whorls are the chief architectural feature of this tumor. Note the invasion of the epidural fat. Hematoxylin eosin stain. ×110.

Another characteristic and frequent architectural formation is the whorl (Figs. 131, 132). This is a spherical or oval structure composed of concentric layers of cells varying from four to a dozen rows or more (Fig. 134). The cells are like those previously described.

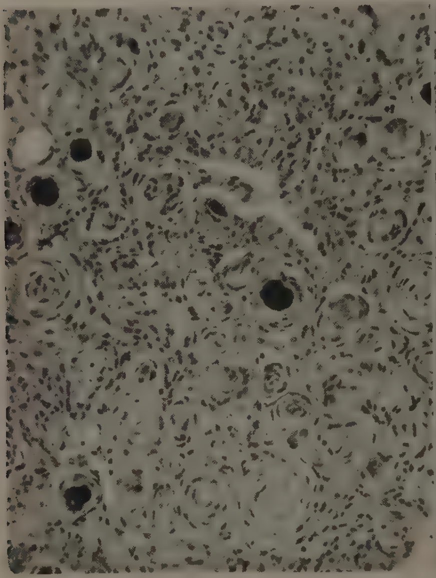


FIG. 132

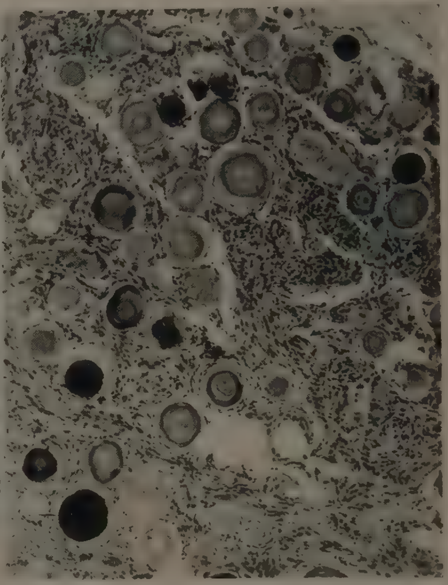


FIG. 133

FIG. 132. Meningioma. The neoplasm was intradural and extended from T 2 to T 3. The tumor is composed of small whorls some of which show central degeneration with resulting fibrosis and hyalinization. Some of the latter are partially calcified, giving rise to psammoma bodies. Hematoxylin eosin stain. ×230.

FIG. 133. Meningioma. The neoplasm was intradural and extended from T 2 to T 3. Many whorls are in various stages of calcification resulting in the formation of psammoma bodies and there is a considerable reduction in the number of well preserved tumor cells. Islands of tumor cells resemble arachnoid granulations. Hematoxylin eosin stain. ×110.

The central cells are polygonal or rounded, while the enveloping cells in the concentric layers are elongated and often progressively narrower and more slender in each succeeding layer from within outward. Frequently, fine reticulin and collagen fibers are found between the cell layers (Fig. 136). Not infrequently a capillary forms the central structure about which the tumor cells are layered (Fig. 134B). Pericellular and perivascular whorls are present in 52 of the 57 meningiomas, the former predominating markedly. In 14 neoplasms, they are the dominating architectural feature, in 17 abundant, in 12 moderately frequent, and in 6 scattered and occasional. The whorls may be closely approximated or separated by one or two rows of cells in slender curving columns (Figs. 132, 133) or, more rarely, by a delicate band of connective tissue fibers.

Progressive degeneration with fibrosis, hyalinization, and calcification of whorls is common, resulting in the formation of psammoma bodies (Fig. 133). These are small concentrically layered spherical masses of calcium of the size and outline of the whorls from which they arise (Fig. 135). The degenerative process, similar to that which occurs in arachnoidal granulations, progresses from within outward; and the successive fibrosis, hyalinization, and calcification of succeeding layers produce the final concentrically laminated, calcium spherule (Fig. 135). These psammoma bodies are present in 51 of the 57 meningiomas and are abundant or predominating in 25. The tendency to psammoma formation seems to be greater in intraspinal than in intracranial meningiomas. Where the psammoma bodies are

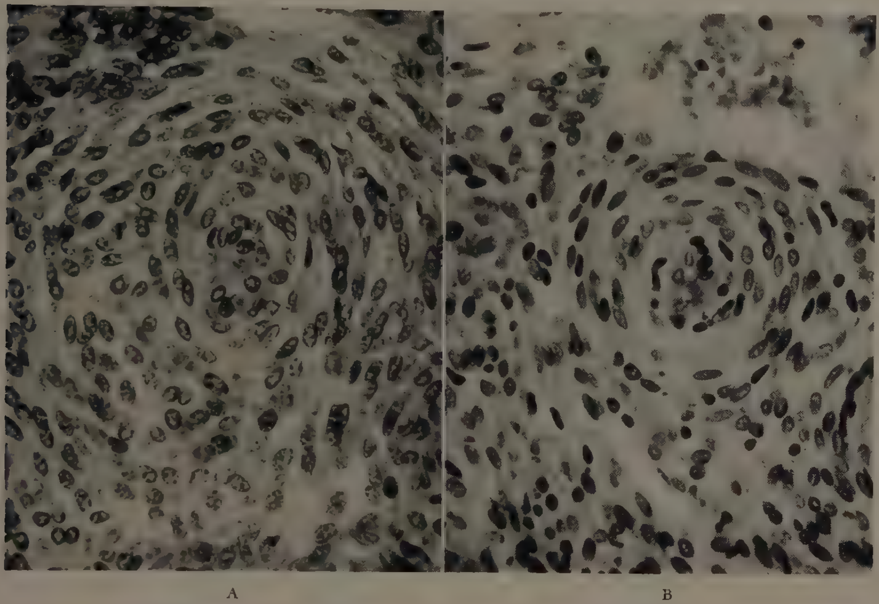


FIG. 134. A. Meningioma. The neoplasm was intradural at T 7. This is a whorl consisting of concentric layers of tumor cells about a central cluster of such cells. The center is double and is composed of poorly outlined polygonal cells. About these are the elongated elements. Note the uniform, oval, light-staining nuclei so characteristic of the cells of this tumor. Hematoxylin eosin stain. ×460.

B. Meningioma. The neoplasm was intradural and extended from T 2 to T 3. This is a perivascular whorl consisting of concentric layers of elongated tumor cells about a capillary. Hematoxylin eosin stain. ×460.

particularly abundant, and this is most striking in 6 of the tumors, the intermediate cell columns, clusters, and whorls are compressed or completely atrophic and are replaced in part or entirely by relatively acellular, dense fibrous tissue consisting of collagen and reticulin fibers. In such areas, too, the psammoma bodies have a tendency to fuse and form irregular calcium spicules.

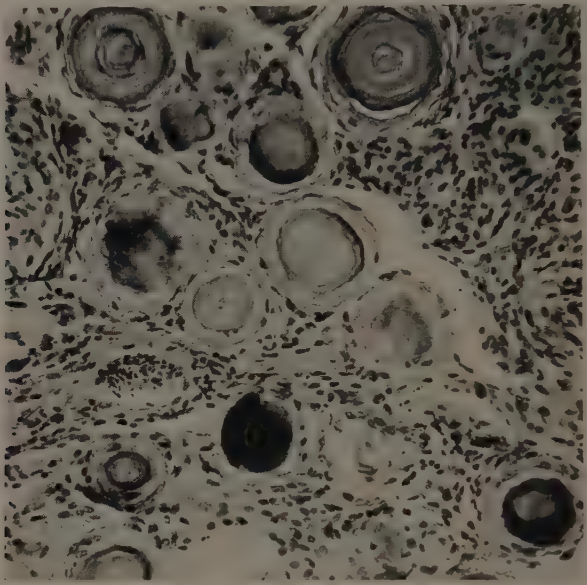


FIG. 135. Meningioma. The neoplasm was intradural and extended from T 2 to T 3. Whorls in various stages of concentrically laminated calcification are being converted into psammoma bodies. Hematoxylin eosin stain. ×300.

Connective tissue fibers are scanty in all but 5 of the 57 meningiomas. In 33, collagen and reticulin fibers are confined to blood vessel walls, to some of the whorls (Fig. 136) and to scattered narrow, vascular connective tissue trabeculae. In 11 neoplasms, the major portion of each tumor contains as little fibrous tissue as do the 33 tumors in the first group referred to. In some foci in these tumors, however, where there are many psammoma bodies, there is considerable fibrosis. Medium-sized and broad connective tissue trabeculae also traverse some portions of the tumor, dividing it into lobules. In some areas, these are very numerous and are connected by many smaller trabeculae, compressing the intertrabecular tumor cell masses and producing atrophy in them. Connective tissue fibers may occasionally be present over a considerable area between parallel columns of compressed but preserved tumor cells. The general architecture of the area is preserved but the individual cells are more slender, spindle-shaped, and their nuclei often darker. These connective tissue trabeculae appear to be of dural origin and seem to be most abundant in those portions of the tumor nearest its dural attachment. In 6 of the tumors, fibrosis is extreme, and only occasional islands of typical tumor cells, whorls, and numerous psammomata embedded in the dense mass of collagen and reticulin give evidence of the true nature of the tumor. In general, however, the paucity of connective tissue fibers in these tumors is in contrast to their abun-

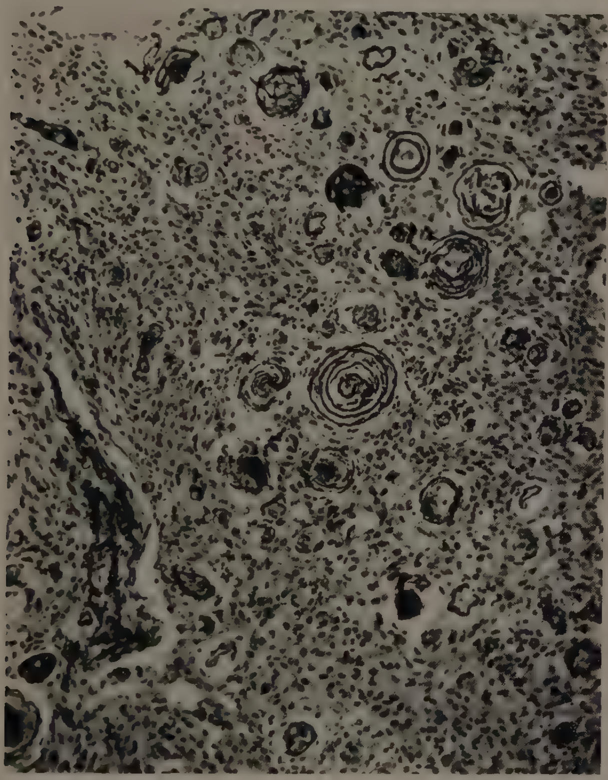


FIG. 136. Meningioma. The neoplasm was intradural and extended from T 2 to T 3. Reticulin fibers are confined to walls of blood vessels and to some whorls where they follow the concentric lamination of those structures. Foot-Bielschowsky stain. ×110.

dance in the solid zones of nerve sheath tumors which may resemble their architecture in part.

The major portion of each of these meningiomas is only slightly vascular. The greatest vascularity is in the capsule. In the rest of the tumor, the vessels are confined to the fine and heavier connective tissue trabeculae and occur in the centers of some of the whorls. In the fibrotic areas they are often secondarily concentrated and show abnormal changes in their walls, such as fibrosis, thickening, and hyalinization with a reduction in the size of their lumina.

Like the intracranial meningiomas, the spinal meningiomas may be locally invasive. Two of the extradural neoplasms in this series invade the epidural fat (Fig. 131); invade the dura extensively (probably a great many more do so to a minor degree); in 2 instances there is evidence of invasion of the vertebrae in which the invaded marrow spaces are completely fibrosed and the bony trabeculae atrophic; and in one instance the spinal cord is superficially invaded, although this was not verified histologically. The less intimate connection between spinal dura and vertebrae, as compared with that of the intracranial dura and skull, probably accounts for the comparatively rare occurrence of vertebral invasion as compared with invasion of the cranium. Invasion of the spinal cord is equally rare or rarer. Antoni (1936) could be sure of only one reported case, that of Forster (1913).

Sheets of tumor cells, such as were seen in 3 of our neoplasms, have been described by Bailey and Bucy as characteristic for their meningeal tumor, meningotheliomatous type; and they occur also in Globus' primitive meningioma, and in the meningioma Type I, Variant 1 of Cushing and Eisenhardt. The curving columns of parallel cells, the cell islands (of the type of arachnoidal granulations), whorls and psammomata occur in Bailey and Bucy's meningeal tumor, psammomatous type; in Globus' leptomeningioma; and in the meningioma of Cushing and Eisenhardt, Type I, Variants 2-4, and Type II, Variants 1-4. Fibrosis, diffuse and trabecular, has been described in tumors classed as meningeal tumor, fibroblastic type, by Bailey and Bucy; in the pachymeningioma and leptomeningioma, by Globus; and their meningioma, Type III, Variants 1-3, by Cushing and Eisenhardt.

It seems from our own material that the cells in the infrequent meningiomas showing fibrosis are not fibroblasts, as is implied above, but cells of exactly the same type seen in our other meningiomas, which have, however, undergone regression due to compression. Typical structures of the other types may be found in the nonfibrotic areas, and the architecture in the fibrotic zones, when not too much distorted, is still easily recognizable as being fundamentally the same as in the nonfibrotic zones.

Ossification has been described by Bailey and Bucy as occurring in their meningeal tumor, osteoblastic type; by Globus in his primitive meningioma; and by Cushing and Eisenhardt in their meningioma, Type VII (osteoblastic meningioma). No example of an apparently undifferentiated type of mesodermal tumor, referred to by Bailey and Bucy and by Globus as mesenchymal type of meningioma, was encountered in our material.

It seems to us that although one or another of the structural types or tissue components may predominate, they often occur together, and they may be associated in many combinations. The essential unity of these neoplasms, however, remains and the uncomplicated term meningioma would seem adequate and would avoid a complicated terminology.

The angioblastic meningiomas are rare and will be treated with the closely related hemangioblastomas. Melanoblastomas (meningeal tumor, melanoblastic type, of Bailey and Bucy; pial melanoblastic meningioma of Globus), lipomas (meningeal tumor, lipomatous type, of Bailey and Bucy; lipoblastic meningiomas of Cushing and Eisenhardt), chondromas and osteosarcomas (chondroblastic meningioma of Cushing and Eisenhardt) will be treated among the rare intraspinal neoplasms. Although some do arise and others may arise from the meninges, it would seem wise to delimit the term meningioma to one rather well defined and common neoplasm.

At first, meningiomas were thought to be cancerous growths of the meninges (Cruveilhier et al, 1829). Virchow (1847) was convinced that they were sarcomas of the dura and named those which contained many fine calcium particles, "brain-sand," psammomas. Lebert (1851) also referred to them as fibroblastic tumors. The term epithelioma was then proposed (Meyer, 1859; Bouchard, 1864; and Robin, 1869) on the basis of some of the tumor's epithelial-like structures and its origin from the supposed epithelial lining cells of the meninges. Golgi (1869) following His' designation of such lining cells as endothelial, suggested endothelioma. Although most of those mentioned considered these tumors to be of dural origin, Robin (1869) thought that they arose from the arachnoid. Cleland (1864), a few years earlier, in describing two such neoplasms as tumors of the arachnoid, derived them from arachnoid granulations. Schmidt (1902) demonstrated that the histological appearance of meningiomas was not only similar to that of arachnoid villi and granulations but to the arachnoidal cell nests implanted in the dura. Aoyagi and Kyunō (1912), studying the dura of individuals of various ages, determined the sites of predilection for arachnoid cell clusters, compared them with the sites of occurrence of tumors (evidently meningiomas), found them to correspond approximately and concluded that the tumors arose from the cell clusters. Cushing and Weed (1915) pointed out that the calcific and osseous deposits in the arachnoid were similar to those in meningiomas. Weed (1920) demonstrated gradations from enlarged arachnoid granulations in the cat to probable tumor formation. Cushing (1922) strongly supported these views and confirmed the findings of Aoyagi and Kyunō by mapping the sites of occurrence of a series of intracranial meningiomas.

Oberling (1922) postulated the theory that the leptomeningiomas were of neuroectodermal origin, and meningeal tumors began to be spoken of as neuroepitheliomas, gliomas, and the like, by French pathologists. The work of Harvey and Burr (1926) adduced experimental evidence to support this theory of the origin of the leptomeninges, while that of Flexner (1929) contradicted it. Harvey, Burr and Van Campenhout (1933) and later Raven (1936) again brought forward evidence of the neural crest theory of the origin of the leptomeninges. Whatever may finally be generally agreed upon, it is obvious from their structure and reactions that when fully developed, the leptomeninges are connective tissue in type. That such may be the case, though their origin be ectodermal (neural crest), is supported by the work of Platt (1893-97), Stone (1921-29) and more especially Vogt (1929) in which the sharp demarcation between the germ layers and their products is discounted. The tumors of the leptomeninges may, therefore, properly be looked upon as connective tissue tumors.

The original observations of Cleland and Robin, the illuminating work of Schmidt, the excellent evidence gathered by Cushing and Weed, and the corroborative evidence offered by the findings of Aoyagi and Kyunō, all point definitely to the origin of meningiomas (as delimited here) from arachnoidal granulations. The cells (particularly the nuclei) of arachnoidal granulations are exactly like those of the meningioma. The similarity of the architectural formations, in the arachnoid granulation and in the meningioma; the concentric layering corresponding to the whorl and the tendency to proliferate in sheets, island clusters and columns often connected by cellular bridges seen in each; the tendency for whorls in each to undergo exactly similar degeneration with the formation of psammoma bodies; the occasional tendency to diffuse fibrosis with strangulation of the cells with increasing age of the granulation or neoplasm, and the occasional ossification in each; and finally, the regular occurrence of meningiomas at those sites at which arachnoidal granulations are most abundant and at the ages at which they are most richly proliferative, all serve to support this view.

That meningiomas are so regularly attached to dura is due to the regular invasion of the dura by arachnoid granulations in the later decades of life, their proliferation in situ after attenuation and separation of the pedicle attaching them to the arachnoid, and the origin of the meningiomas from such dura-implanted clusters. The meningioma capsule and the heavy connective tissue trabeculae seen in some meningiomas are probably dural in origin and depend upon the initial growth between the dural lamellae.

As has been shown by Odyniec (1908), Bluntschli (1908), Elman (1923), and Hassin (1930), arachnoidal cell clusters, villi, and granulations occur in the spinal canal at the points of emergence of the nerve roots where the arachnoid membrane is in contact with the dura. The villi invade the spinal dura as they do the cerebral. Here psammoma bodies are also found. That the spinal meningiomas like the cerebral arise from such arachnoidal cell nests seems most likely.

GLIOMA

The gliomas of the spinal cord like those of the brain arise predominantly from the interstitial elements of the neural parenchyma. Until the latter were more fully understood, following the investigations of Cajal and Hortega, the nature of many of the gliomas was rather obscure. Using the methods of these investigators, Bailey and

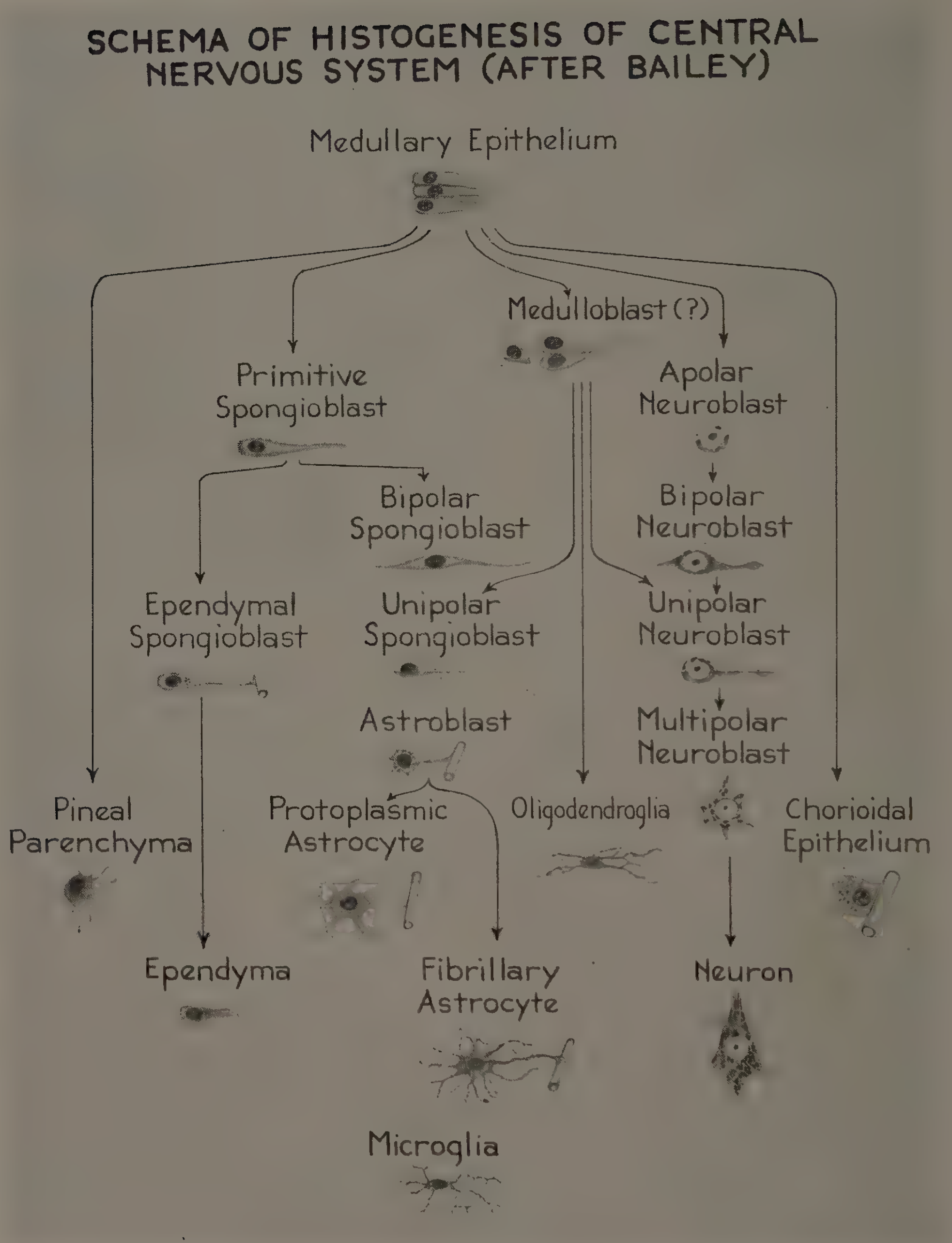
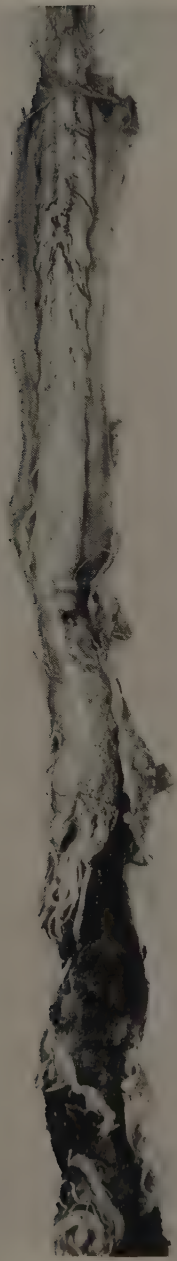


FIG. 137. Schema of histogenesis of central nervous system. (After Bailey.)

Cushing were able to identify many of the cell types in gliomas of the brain, and to classify these tumors on the basis of their chief cell types as well as their structure. In their pioneer classification, an attempt was made to identify tumors, not only by adult cell types, but also by attempting to detect resemblances to the embryological and developmental forms which preceded them (Fig. 137). This has proved to be a very fruitful approach and although it has been repeatedly found necessary to modify many of the original conceptions of this classification, a modification participated in by its originators, it has withstood the test of use and is eminently suitable as the basis for a systematic consideration of spinal cord gliomas.

It has become general usage to include under the term glioma, those neoplasms arising from the parenchyma which are made up in part or wholly of ganglion cells or their precursors. It would seem wise for the sake of simplicity to continue this usage—although these tumors are not in a true sense gliomas, since they are made up wholly or in part of neural elements—rather than to employ a special term, such as paraglioma which has been proposed by Hortega (1932) and Henschen (1934). This applies also to the choroidal and pineal tumors.

The divergency of conception, classification, and nomenclature renders the early literature on spinal cord gliomas of little use in attempting to determine the characteristic type, location, and other features of these tumors. It is difficult to be certain from the isolated reports of such lesions just what type of tumor is being described since in the great majority of instances, the descriptions have preceded the more recent investigations on the interstitial cells of the nervous system. It is difficult, therefore, to reclassify them although Bailey and Foerster (1936) made a rough attempt to do so with 92 reported instances. Kernohan, Woltman and Adson (1931) were the first to study a large group of spinal cord gliomas, applying the classification of Bailey and Cushing, and Kernohan and various associates have steadily added to these data (1932, 1933, 1940). In their first report they presented a group of 51 intraspinal tumors, among which were 42 gliomas; in their latest communication (Rasmussen, Kernohan and Adson, 1940) this group had grown to 95. Elvidge, Penfield and Cone (1937) subjected a collection of 20 intraspinal gliomas to a similar analysis in studying 210 gliomas of the central nervous system. Bailey and Foerster (1936) classified eight spinal tumors of this type. These three series added



to our own group of intraspinal gliomas will form the basis of some generalizations.

Of the 253 intraspinal neoplasms in our series, only 26 are primary gliomas, or approximately 10 per cent. Rasmussen, Kernohan and Adson (1940) found 95 gliomas among 557 verified intraspinal neoplasms, or 17 per cent. These investigators did not include metastatic carcinomas in their total and the difference between the two series may depend in part on this discrepancy.

Ependymoma

Ependymoma (ependymoblastoma, épendymocytome, glio-epithelioma, etc.). This is a tumor composed essentially of adult ependymal cells (Fig. 137). As has been noted previously, this is the most common glioma of the spinal canal. In our small series of 26 gliomas, there are 20 ependymomas. Of these, 14 are in or reach the lumbar sac, lying in relation to the conus medullaris or the filum terminale or both, and the cauda equina. Four lie wholly within the lumbar sac; 3 are related to the filum and one to the conus and filum. Five ependymomas fill or almost completely fill the lumbar sac and extend upward into and around the sacral and lumbar segments of the spinal cord (Fig. 138). One of these tumors extending into the lumbar subarachnoid space rotated the spinal cord in this region through

FIG. 138. An ependymoma which displaced and compressed the cauda equina and filled the dural sac to distension. The sacral, lumbar and thoracic segments, up to the third thoracic, are enlarged and pallid. The lower segments show the greatest swelling and are distorted; the lumbar segments are rotated clockwise. The tumor about the cauda equina is deep red, due to its vascularity and to hemorrhages into it. There is gross invasion of the lumbar subarachnoid space.

an angle of almost 90 degrees (Fig. 139). The cord is swollen but nowhere invaded by tumor; in the upper lumbar segments there is a syringomyelia in the posterior columns and commissures which appears to be situated asymmetrically because of the rotation of the cord (Fig. 151). Although the central canal is still patent, many buried ependymal cell clusters form a garland around it and appear to have proliferated (Fig. 150). It is from such cell groups in the filum terminale that this tumor probably arose. Tumor implants in the subarachnoid space are found as high as the third thoracic segment and the cord is rotated as high as the sixth thoracic segment (Fig. 139). This case will be referred to again in the section on implantation of gliomas in the subarachnoid space and in that on spinal cord tumors and syringomyelia.

Five of the ependymomas have their caudal extremity in relation to the uppermost part of the filum and to the conus, and extend upward into and about the sacral and lumbar segments of the spinal cord (Fig. 139), while one begins at the level of the conus and extends upward through the sacral segments. Two of the ependymomas occupy the lumbar and sacral cord segments, 2 are lower thoracic and upper lumbar, one is

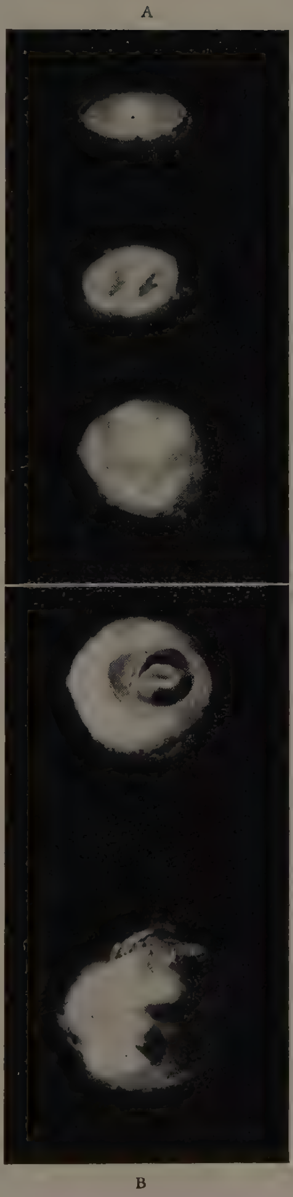


Fig. 139. The dark mass in the subarachnoid space to the right of the fifth lumbar segment of the spinal cord is an ependymoma with hemorrhages into, but no invasion of the subjacent parenchyma. The spinal cord is markedly rotated and swollen; the architectural markings are obscured. There is a syrinx in the fourth lumbar segment containing gelatinous material and having a grayish glial wall. The rotation of the spinal cord is still visible in the second lumbar and sixth thoracic segments, with obscuration of the architectural markings and edema and retraction of the gray matter. Note the subarachnoid tumor on the right side of the second lumbar segment.

lower thoracic, and one is a high cervical tumor and extends into the posterior cranial fossa.

The ependymomas form a little over three-fourths of our series of 26 intraspinal gliomas. Of the 95 intraspinal gliomas of Rasmussen, Kernohan and Adson, 65 were ependymomas, or 67 per cent. Of these, 6 occurred in the cervical spinal canal, 26 in the thoracic region; 29 in the lumbar region, and 4 in the sacral region anterior to the coccyx. Of our 20 ependymomas, 4 are extramedullary (lumbar sac), 12 are intra- and extramedullary, and 4 are intramedullary. One of the extramedullary tumors in the lumbar sac is both intra- and extradural. Rasmussen, Kernohan and Adson listed 33 as intramedullary and 32 as extramedullary, and subdivided the latter into 27 intradural, and 3 intra- and extradural (from the intradural portion of the filum terminale), and 2 extradural (from the extradural portion of the filum terminale).

Among the 784 histologically verified cerebral gliomas in our records, only 23 are ependymomas. Of a total of 43 ependymomas of the central nervous system, 20 are from the spinal canal.

Elvidge, Penfield and Cone (1937) in studying 210 verified gliomas of the central nervous system, found 190 to be intracranial and 20 intraspinal. Of 19 ependymomas in this total series, 11 were intracranial and 8 intraspinal. Kernohan and Fletcher-Kernohan found that of 109 ependymomas, 54 were in the spinal canal, while 52 occurred in the cranial cavity, and 3 were presacral. Bailey and Foerster (1936), in reviewing 92 intraspinal gliomas gathered from the literature, to which they added 8 instances of their own, identified 39 as ependymomas. When one considers the much rarer occurrence of gliomas in general in the spinal canal, the predominance of spinal ependymomas is quite striking.

The ependymomas are reddish, purplish or brownish red, or grayish in color. Usually they are quite soft in consistency, although occasionally they may be fleshy or tough; rarely they are firm, resistant, and almost cartilaginous. Their surfaces may be granular, irregular, or smooth; and when they lie in an extramedullary, intradural location (usually in the lumbar sac), they are ordinarily covered by a thin delicate connective tissue membrane derived from the leptomeninges (Fig. 138). One of the 20 ependymomas contained grossly visible cysts which were smooth-walled, small, lay within the tumor substance, and were filled with clear yellowish fluid.

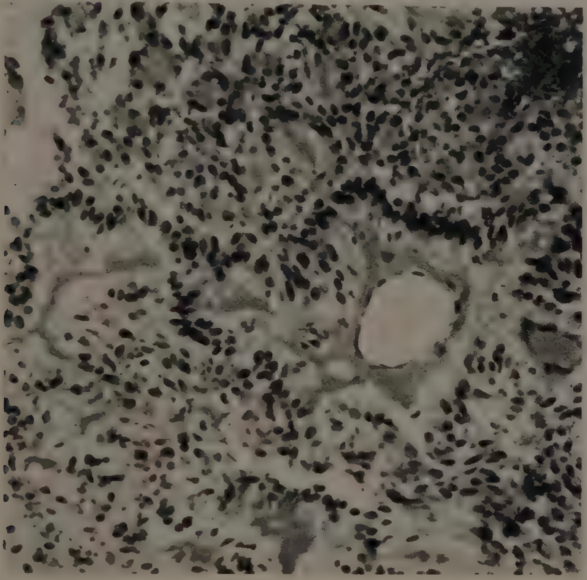


FIG. 140. Ependymoma. The tumor occupied the region of the cauda equina and the spinal cord from L 5 to S 2. Slender columnar cells are arranged radially about blood vessels of the order of capillaries. The bodies of the cells are attached to the vessel walls and are often attenuated. The nuclei are oval, hyperchromatic, and at the extremity of the cell distal from the vessel. Hematoxylin eosin stain. ×300.

Kernohan, Woltman and Adson (1931), in their study of 51 spinal cord tumors (42 gliomas), described three varieties of ependymoma. Kernohan (1932) designated these as epithelial, cellular, and myxopapillary. Kernohan and Fletcher-Kernohan (1937), in a histological analysis of 109 ependymomas, were able to distinguish four types: myxopapillary, epithelial, and cellular ependymoma, and choroid papilloma. Although the last tumor had been listed as a separate entity in the classification of Bailey and Cushing (1926) and of others subsequently, the Kernohans felt that since the choroidal cell is simply a specialized ependymal cell, the tumors which it forms should be included with the ependymomas. In the spinal canal, of course, this last type of tumor cannot be expected to occur. As has been pointed out by Kernohan, Woltman and Adson (1931), it is unusual to find a neoplasm in which the architecture is limited to one of these types; and the Kernohans (1937) stated that only a small number of tumors are purely of one type. Often two and occasionally all three types of architecture are represented in each of our ependymomas. Tarlov (1934) who studied a small ependymoma of the filum terminale in serial sections and found the pattern varying from section to section, and even within a single section, suggested that the subdivision of ependymomas into various pattern types was not advisable. Bailey (1938) pointed out that the ependymomas described as myxopapillary by Kernohan et al., are really pseudopapillary, due to intervascular degeneration which he observed in a number of such neoplasms. This observation was confirmed in our material in which typical perivascular cell groups were secondarily separated by an apparently chronic degenerative process in the intervascular tissue with a resultant pseudopapillary appearance. Under the circumstances, it would seem simpler to recognize these variations in the architecture of what is essentially a single tumor type without further subclassification, and to refer to these neoplasms only as ependymomas (with the exception of the choroidal papilloma which does not further enter into this discussion of spinal cord gliomas).

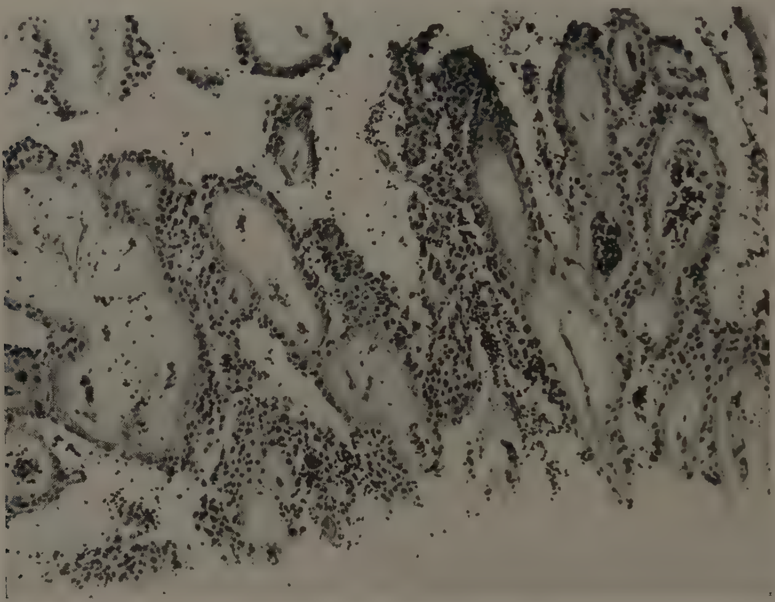


FIG. 141. Ependymoma. The neoplasm was intramedullary and extended from T 12 to S 1. Note the perivascular arrangement of the tumor cells and the papillary or pseudopapillary architecture. There is a myxomatous change in the walls of the blood vessels and the perivascular connective tissue, displacing the tumor cells radially away from the vessel lumen. Hematoxylin eosin stain. ×110.

Histologically, the neoplastic tissue forms a continuous, fairly cellular, highly vascular sheet, which in some zones may become loose-meshed, pseudopapillary, and show some of the degenerative features described below. These two may be mingled in any proportion, although the densely cellular areas predominate most often.

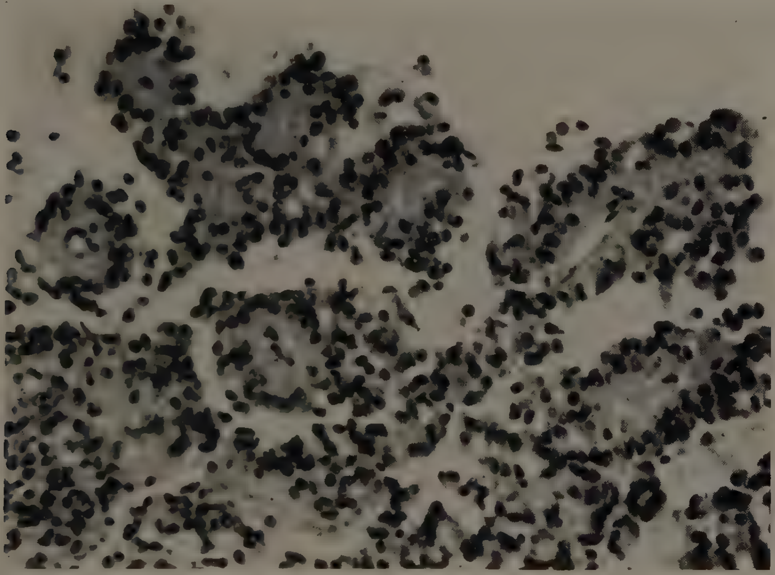


FIG. 142. Ependymoma. There is a perivascular arrangement of cuboidal and columnar cells attached by a broad cytoplasmic base to the walls of the capillaries. The architecture is papillary or pseudopapillary. Hyperchromatic nuclei are eccentrically placed at the end of the cell farthest from the blood vessel. Hematoxylin eosin stain. ×140.

Scattered through the cellular zones are varying-sized, usually small, rosettes of cells which may cluster in some areas, but which only infrequently form the predominant architectural feature, as may either one of the two other patterns.

In the highly vascular, cellular zones, a great many of the tumor cells are found radially arranged about blood vessels, chiefly capillaries (Figs. 140, 141, 142). These cells are low or high cuboidal, more often columnar or high columnar (Figs. 143, 144), and bulbous, pyriform, fish-tail or irregular in shape and elongated (Figs. 145, 146). They lie closely approximated with their long axes at right angles to the vessel wall and one extremity of the cell resting upon or abutting against it (Fig. 144). At times, the cells can be seen to form a continuous limiting membrane resting on the vessel wall or secondarily retracted from it (Fig. 146). The portion of the cell nearer the blood vessel may undergo varying degrees of attenuation, resulting in a slender, straight or deflected, curving or twisted extension to the vessel wall (Figs. 140, 145, 147). The cytoplasm of these cells is finely or coarsely granular and acidophilic and the cell boundaries are sharp (Fig. 143). The cells have no protoplasmic processes or fibers and contain no neuroglial fibrillae. The nuclei are large, oval, fairly light-staining, and contain a few coarse chromatin granules (Fig. 144). The nucleus is usually at the far end of the cell from the vessel. In the cytoplasm adjacent to the nucleus, on the side toward the vessel or on the opposite side, there is often a group of short rods and granules which stain by the ethyl violet-orange G or phosphotungstic acid hemotoxylin stain and are known as blepharoplasten. These are not always easy to detect and may occur scantily in some of the more cellular areas. Occasionally translucent zones or vacuoles may occur in the cytoplasm, especially on the side of the nucleus nearer the vessel wall (Fig. 144).

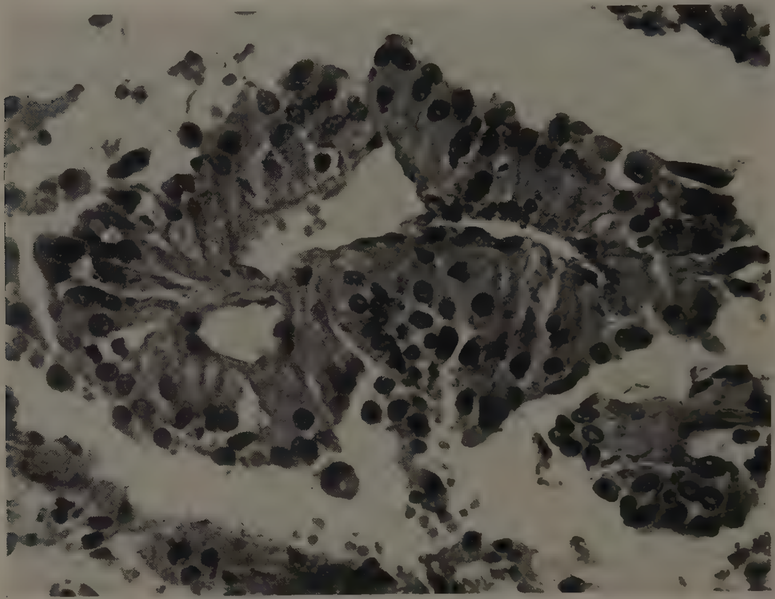


FIG. 143. Ependymoma. The neoplasm involved the cauda equina and spinal cord between L 5 and S 2. Slender, columnar, ependymal cells are arranged about the capillaries. The nuclei are hyperchromatic, spherical or oval and lie distal from the vessel wall. ×460.

When the attenuation of that part of the cell cytoplasm resting upon the vessel wall becomes extreme, the tumor tissue may take on quite a loose-meshed appearance and the vascular attachments are not always obvious (Fig. 147). In such areas, one frequently encounters apparent intervascular islands of cells in which there is nuclear clustering and the cytoplasm is scanty, although the elongated form of the cell can be made out (Fig. 149). Comparison, however, indicates that these are probably groups of cells related to some vessel out of the plane of section and transected through that perinuclear portion of the cytoplasm furthest removed from the vessel. Areas of slow degeneration and disappearance of tumor cells may occur in such loose-meshed zones, resulting in the formation of small cysts (Fig. 149). Occasionally, as in one of our tumors, these may become macroscopic.

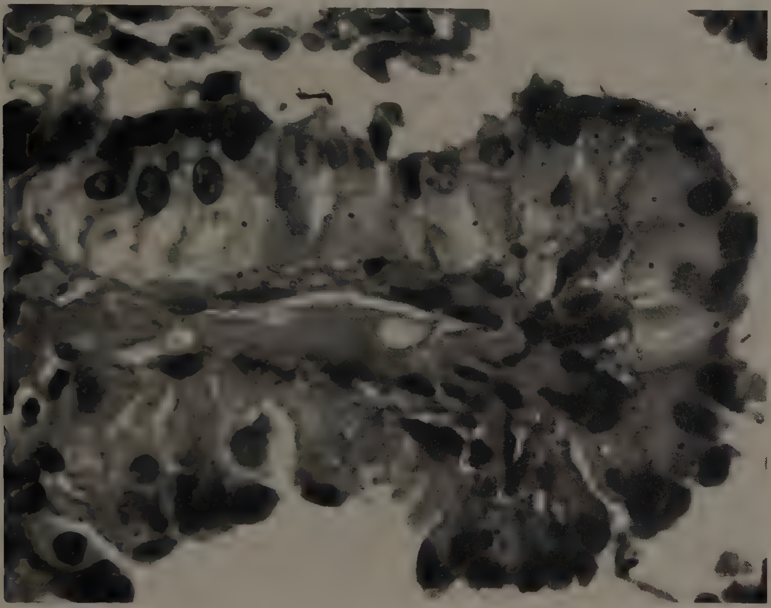


FIG. 144. Ependymoma. The tumor occupied the region of the cauda equina and the spinal cord from L 5 to S 2. The tumor cells are typically columnar. The nuclei contain both coarse and fine chromatin granules and are eccentric. Note the multiplication of endothelial cells in the wall of the central capillary. Hematoxylin eosin stain. ×600.

In the intervascular areas, one may find single examples, rows, clusters, or large sheets of closely approximated, rounded or polygonal elements with sharp or indistinct outlines and spherical, hyperchromatic nuclei and a pale unstained zone or halo about the nucleus. In 2 of our 20 ependymomas, these occupied a number of large areas.

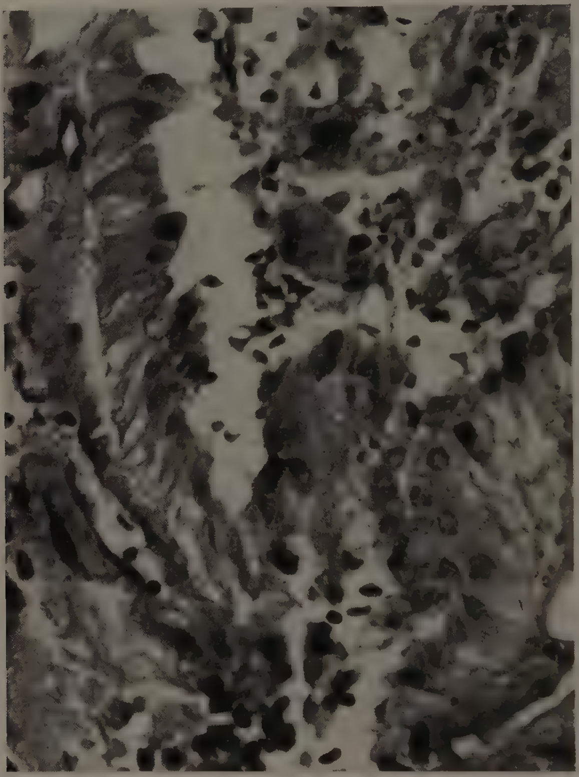


FIG. 145. Ependymoma. The tumor occupied the region of the cauda equina and the spinal cord from L 5 to S 2. The columnar neoplastic ependymal cells show perivascular arrangement. Note the attenuation of the cytoplasmic attachment in some of the cells. Hematoxylin eosin stain. ×600.

The Kernohans (1937) pointed out the frequent occurrence of such cells in the more cellular portions of ependymomas, and considered them to be oligodendroglia, which their appearance certainly suggests. In two instances, the Kernohans were able to stain these by Hortega's silver carbonate stain and demonstrate their oligodendroglial nature. Tarlov (1934) described scattered spongioblasts, astroblasts and astrocytes as well in his ependymoma of the filum terminale. Such an intermixture of other cells of neuroectodermal origin in a tumor predominantly composed of cells of one type is not surprising considering their common origin, and occurs frequently since oligodendroglia can be found in some astrocytomas, astrocytes, in many ganglion cell tumors, and so on.

In some areas there may be degeneration of intervascular elements and of some of the most elongated of the vascular oriented cells (Fig. 149), as noted in the description of the formation of small cysts. As Tarlov (1934) has pointed out, fat may be found in tumor cells and in occasional large mononuclear phagocytes. The islands of radially arranged perivascular neoplastic elements then stand out sharply as the intervascular cellular debris is cleared away, resulting in what Bailey (1938) calls a pseudopapillary appearance (Figs. 141, 142). The degeneration is evidently slowly progressive and no massive areas of necrosis are seen. There is an increase in the number of subendothelial connective tissue fibers about the central capillary of the perivascular formations, which may go on to considerable pericapillary fibrosis. This compact band of relatively acellular connective tissue frequently becomes edematous, swollen, and undergoes mucinous degeneration (Figs. 140, 141, 146). Mucin can readily be stained in the cores of these papillae or pseudopapillae.

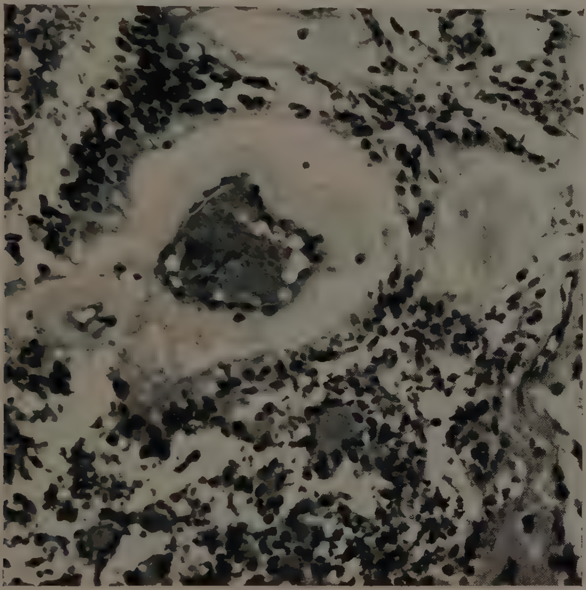


FIG. 146. Ependymoma. The tumor occupied the region of the cauda equina and the spinal cord from L 5 to S 2. There is a myxomatous change in the walls of a blood vessel with displacement of the radially arranged perivascular tumor cells to a distance from the vessel lumen. Note the small true rosettes containing dense homogeneous material in their lumnia. Eosin methylene blue stain. ×300.

A predominance of this tumor pattern in some ependymomas is the basis for the subgroup of myxopapillary ependymomas of Kernohan. Occasionally the central capillary may show endothelial hyperplasia (Figs. 144, 146). At times a marked increase in the pericapillary fibrous tissue may be followed by its partial or complete hyalinization. The perivascular elements may be displaced to a considerable distance from the central vessel lumen and rows of cells of adjacent perivascular formations may be approximated and compressed. The perivascular orientation of the cells is not always easily detected in such areas.

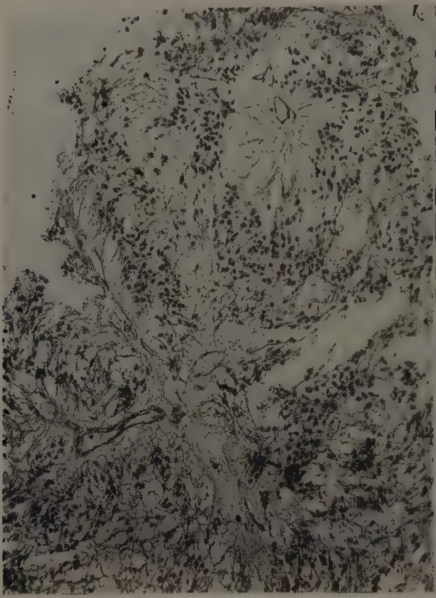


Fig. 147

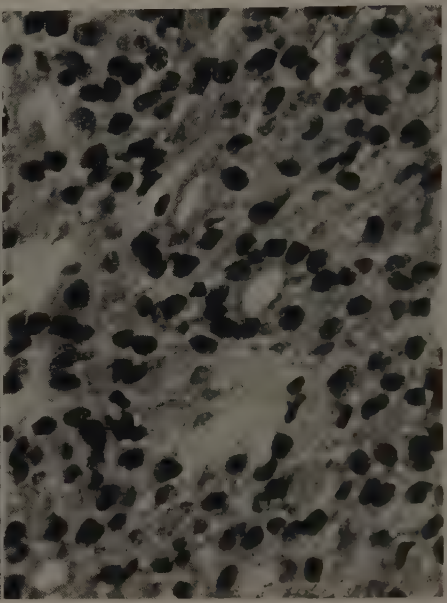


Fig. 148

Fig. 147. Ependymoma. The tumor lay in the fourth ventricle and in the spinal canal from C 1 to C 3. Myxomatous area. The neoplastic tissue is markedly loose-meshed. Cytoplasmic attachments of perivascular tumor cells are extremely attentuated and such connections are often obscured. Mucinoid material is present interstitially. The walls of the blood vessels show mild myxomatous transformation. Hematoxylin eosin stain. ×110.

Fig. 148. Ependymoma. The neoplasm was intramedullary in the cervical spinal cord. Low columnar and cuboidal cells are arranged to enclose a space and form a true rosette. Hematoxylin eosin stain. ×460.

The architecture of these papillary or pseudopapillary areas is compared by the Kernohans to that of choroid papillomas from which they distinguish it by the very slight amount of delicate, perivascular connective tissue in the core of the papillae in the latter, and the observation that mucin is often contained in the choroidal lining cells. There is a resemblance of the ependymoma to the astroblastoma (Fig. 147) as well, but the larger size of the nuclei in the former, the absence of a well-formed vascular sucker-foot, the lack of multiple, stubby cell processes, the failure of deep impregnation of the tumor cells by Cajal's gold sublimate stain, the ordinarily more compact appearance of the tumor, and the presence at times of the structural feature next to be described, the rosette, frequently help to differentiate the ependymoma from the astroblastoma.

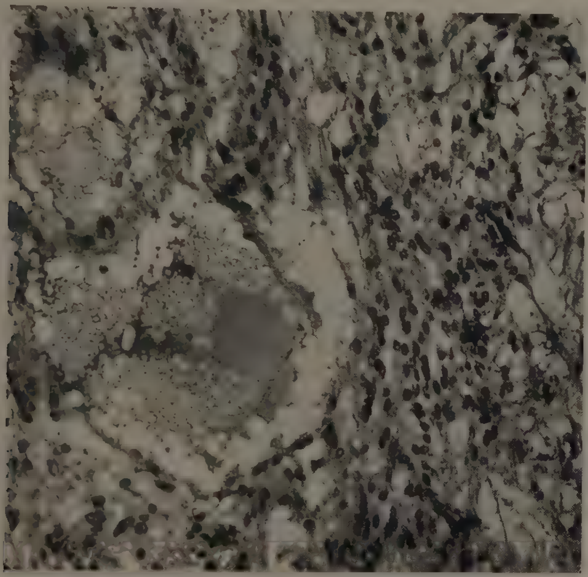


FIG. 149. Ependymoma. The neoplasm occupied the region of the cauda equina and the spinal cord up to T 12. Cellular and cystic intervascular area. Note the elongated and bipolar ependymal spongioblasts with their oval, deeply staining nuclei. Cystic areas, in which tumor cells have degenerated and disappeared contain amorphous material. Hematoxylin eosin stain. ×300.

Occurring singly and scattered, appearing in groups, or rarely dominating whole sectors of an ependymoma are clusters of tumor cells arranged in acini, canals, or rosettes (Fig. 148), occasionally containing dense, homogeneous material (Fig. 146). These resemble the central canal of the spinal cord or those buried clusters of ependymal cells still surrounding a small cavity found at the angles of the ventricles, about the aqueduct of Sylvius and about the patent or obliterated central canal. The resemblance of these rosettes to epithelial structures prompted the Kernohans to speak of the tumors in which they occur in large numbers as epithelial ependymomas (partly the basis, as well, of "glio-epithelioma" for ependymoma of Henschen, 1934).

The cavities are lined by a single layer of cells although at times a multi-layered appearance may result from a varying position of the nuclei. The cells are cuboidal or columnar, sharply outlined, have no cuticular membrane at their free ends facing the cavity, and are not ciliated. Their cytoplasm is granular, acidophilic, not vacuolated, contains no mucus, and nearly always includes blepharoplasten (Fig. 148). The end of the cell away from the lumen either has a basement membrane or is drawn out into a heavy process which is never fibrillar, is quite broad, does not branch, and mingles with the surrounding tumor cells. The Kernohans describe the occasional attachment of such a distal cell prolongation to a vessel, but this was seen only once in our material.

This type of architecture is reminiscent of that of a rare glioma, the neuroepithelioma, a tumor composed of relatively primitive neuroectodermal cells. Bailey (1932) states that it is often difficult to distinguish between some ependymomas and such tumors, and that transitional types may occur. (Roussy, Lhermitte and Cornil, in 1924, described such neuroepitheliomas under the designation épendymogliome.) The deep position of the blepharoplasten in the cytoplasm instead of at the inner border, the absence of cilia, and the deeper staining of the distal prolongation of the cell are given by Bailey (1932) as differential points between the cells lining the rosettes in an ependymoma, and those in a neuroepithelioma. The greater malignancy of the neuroepithelioma (Bailey, 1938) should be taken into account as well. The occurrence in the ependymoma of the other architectural patterns already described serves further to distinguish it.

In all of our 20 ependymomas, there is a mixture of architectural patterns. In 3, the papillary or pseudopapillary architecture dominates most of the tumor; in 5 it comprises about half of the neoplasm; and in the other 13, it occurs in a minor portion of the neoplasm, the compact, well preserved areas predominating. In 3 tumors there is an intermixture of rosettes in the more compact areas.

In the original classification of gliomas by Bailey and Cushing (1926), the ependymal gliomas are divided into ependymoblastomas and ependymomas, the former being composed of immature ependymal cells, ependymal spongioblasts (Fig. 137), and the latter of adult ependymal cells. Later, Bailey (1932) speaking of the ependymal gliomas, says: "They may be subdivided into ependymomas and ependymoblastomas depending on the presence or absence of long tails to the cells, but there seems to be little practical advantage in so doing since both are essentially slowly growing benign tumors." This elimination of the term ependymoblastoma was concurred in by others. More recently, the Kernohans (1937) in investigating a larger amount of material (109 ependymal gliomas), pointed out that one should continue to separate a special group of rapidly growing tumors of the ependymal type. They feel that the term ependymoblastoma should be retained to describe these more malignant neoplasms. In two of the patients having intraspinal ependymomas in our series, the clinical history is approximately three months long as compared to many years in the others. The histology of the tumors, however, is not found to be different from that of the other neoplasms.

The Kernohans (1937) found that of their 109 ependymal gliomas, 59 had well differentiated cells, and 50, cells that were less differentiated. The proportion of these that were intraspinal tumors was not given, however. The total number of intraspinal ependymal gliomas was given as 52. In the report of Rasmussen, Kernohan and Adson (1940), 62 intraspinal ependymal gliomas were listed. The 32 extramedullary (filum terminale) tumors were listed as ependymoma, and the 33 intramedullary neoplasms as ependymoma and ependymoblastoma. The total number of intraspinal ependymoblastomas, therefore, was not clear. Bailey and Foerster (1936) used the terms ependymoma and ependymoblastoma interchangeably in describing two such spinal neoplasms.

The Kernohans, who counciled a retention of this category of ependymoblastoma, described it as follows. The cells are slightly larger than in the ependymoma and the nuclei are definitely larger in proportion to the cytoplasm of the cell, which is slightly basophilic. The cells are rounder, although they may be irregular. Processes are not common. The cells are not usually attached to blood vessels and do not form rosettes. The nuclei are often irregular and hyperchromatic, and mitotic figures are not uncommon. Younger forms of oligodendroglia cells, oligodendroblasts, are present. These are larger than oligodendroglia cells, have sharp cell margins, show some pale, slightly granular cytoplasm in addition to the perinuclear halo, have no processes and have larger, lighter-staining nuclei. Mitoses are common here as well. As has been noted before, no tumor which could be classified as an ependymoblastoma is present among our 20 intraspinal ependymal gliomas.

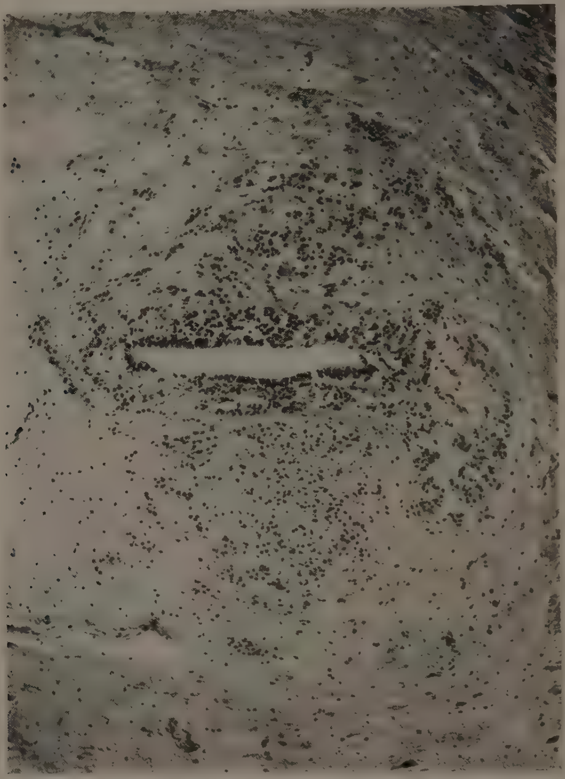


FIG. 150. A section of the lumbar cord in a case of an ependymoma arising from the filum terminale. Note the unusually numerous buried clusters of ependymal cells about the patent central canal. Hematoxylin eosin stain. ×60.

The high incidence of intraspinal ependymomas among the ependymal gliomas of the central nervous system, approximately 50 per cent of the total, both in our material and in that of the Kernohans (1937), requires explanation. These authors advanced a number of very plausible reasons for this circumstance, some of which were previously stated by Kernohan, Woltman and Adson (1933). They pointed out that although the spinal canal is patent throughout in children, it begins to become obliterated shortly after puberty. In the process of obliteration, the ependymal cells are dislodged and dispersed, being spread throughout the commissures as single elements or clusters of cells. Usually a considerable group of them, which often seems greater in number than the total that lined the preexistent central canal and suggests later multiplication, are associated in a cluster of cell nests at the site of the original canal. Occasionally these cells form small secondary canals or rosettes (Fig. 150). This disorganization of the ependymal cells is postulated as a possible explanation of the large number of ependymomas which occur as primary gliomas of the spinal cord.

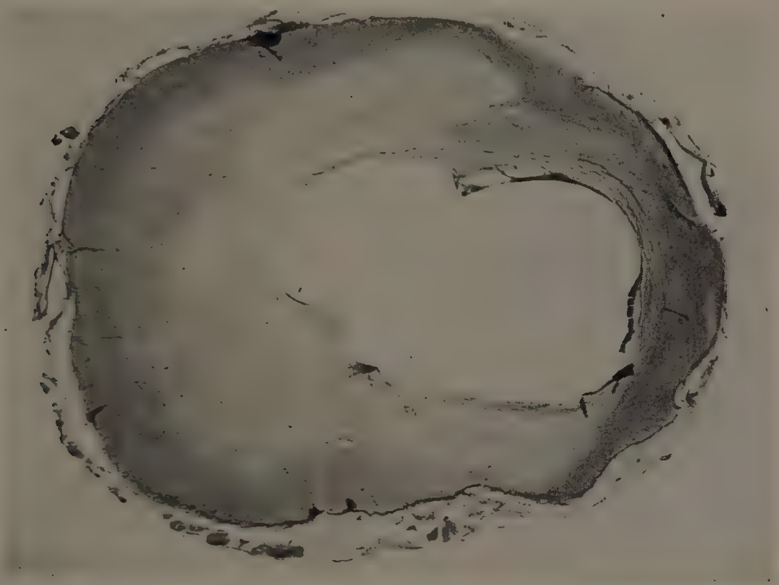


FIG. 151. A syringomyelia in the lumbar cord in a case of ependymoma of the cauda equina region with secondary invasion of the subarachnoid space. Note the rotation of the cord through almost 90 degrees so that the cavity that appears on the right actually lies in the posterior columns.

Of the 20 intraspinal ependymomas in the present series, 9 lie in or occupy the lumbar sac completely. Four of these lie in the lumbar sac and 5 extend into the sacral and lumbar segments of the cord in addition. These burrow among the roots of the cauda, covering them or displacing them to one side (Fig. 138). Five other ependymomas of the 20 extend into the lumbar sac but the major portion of the tumor occupies the spinal cord parenchyma. Rasmussen, Kernohan and Adson (1940) reported 32 such primary extramedullary ependymomas among their 65 intraspinal tumors of this nature.

Kernohan (1924) in his study of the ventriculus terminalis of the central canal found in the lower end of the tapering caudal extremity of the spinal cord, the conus medullaris, demonstrated that the latter contained the same elements as did the spinal cord. While nerve cells, axones, myelin sheaths, astrocytes, and oligodendroglia were less numerous in this structure, ependymal cells were present in large numbers due to the greater size of the ventriculus as compared to the rest of the central canal. Harmier (1933) in studying the intradural filum terminale found that it too contained all the elements present in the cord with the exception of microglia. He noted that many of the neural elements were immature and that a profusion of irregular nests of ependymal cells were distributed in no special order throughout the entire filum. These ependymal elements formed solid islands or small rosettes and the cells lining the latter were ciliated. Tarlov (1934) in serial sections of his filum terminale ependymoma was able to compare the persisting ependymal cell clusters directly with the tumor cells and recorded their resemblance. Kernohan, Woltman and Adson (1933) quite logically derived their gliomas of the cauda equina region from the conus medullaris and filum terminale, although they pointed out that it might be possible that some of these tumors could have originated from heterotopic glial or ependymal masses attached to the nerve fibers of the cauda equina. Of their 25 cauda equina gliomas, 17 were ependymomas. A little less than half of our intraspinal ependymomas and approximately the same fraction of those of Rasmussen, Kernohan and Adson, occurred in this region. The

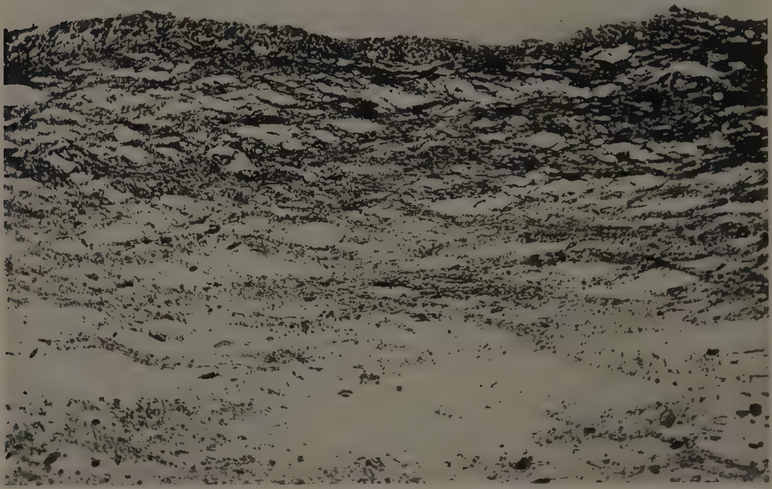


FIG. 152. The glial wall of a syringomyelia cavity in the lumbar cord in a case of cauda equina ependymoma which has invaded the subarachnoid space. Phosphotungstic acid-hematoxylin stain. ×60.

high incidence of ependymomas in this locus may depend upon the profusion and relative predominance of ependymal cells in the conus medullaris and filum terminale.

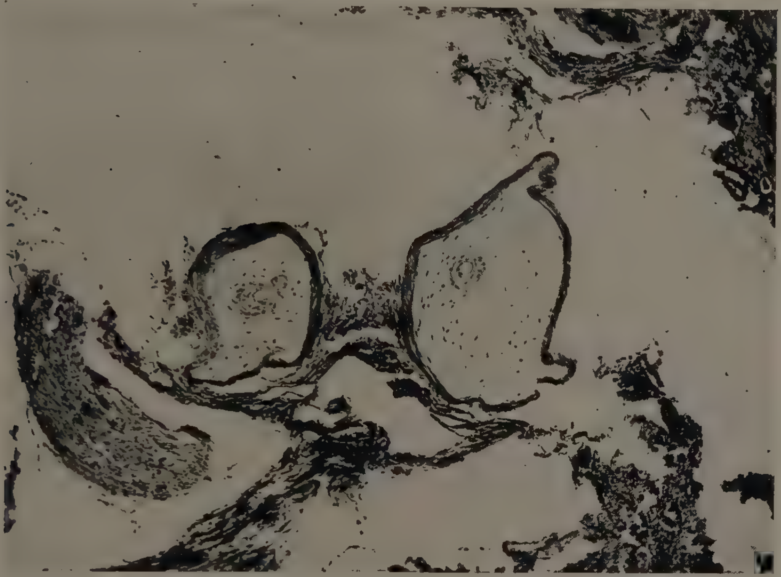


FIG. 153. Leptomeningeal arteries in the lumbar region show obliterative endarteritis in the zone of subarachnoid invasion by an ependymoma of the filum terminale. Hematoxylin eosin stain. ×60.

Although they are not intraspinal neoplasms, brief reference has been made to the ependymomas arising in the hollow of the sacrum. No examples occurred in our series. The Kernohans (1937) described 3 instances among their 109 ependymomas, and pointed out that they are common among the *Mitteldorpf* tumors (those neoplasms occurring in the hollow of the sacrum). They suggested that these ependymomas might arise from the coccygeal medullary vestige which is a remnant of that portion of the neural tube which Kunitomo (1918) and Streeter (1919) have shown extends into the tail bud in early embryonic life.

ASTROCYTOMA

As Bailey (1932) pointed out, the astrocytoma is the classical glioma which was distinguished from other tumors of the neural parenchyma referred to en masse as sarcomas, by the occurrence in it of star-shaped cells (Golgi, 1875). This tumor is composed essentially of the adult glial cells known as astrocytes (Fig. 137).

Of the 26 intraspinal gliomas in our series, 5 are intramedullary astrocytomas.

In Kernohan's (1932) group of 67 intraspinal gliomas, he listed 8 astrocytomas. Among the 95 intraspinal gliomas of Rasmussen, Kernohan and Adson (1940), 10 intramedullary tumors were grouped as astrocytomas and polar spongioblastomas (the number of each not specified), and 6 neoplasms were designated as extramedullary astrocytomas. Among 20 intraspinal gliomas of Elvidge, Penfield and Cone (1937), there were 2 astrocytomas. Kernohan, Adson and Woltman (1933) found one such tumor among 25 gliomas in the region of the cauda equina. In sharp contrast, is the high incidence of astrocytomas among intracranial gliomas, where it forms the largest single group. Among Cushing's (1932) group of 687 classified intracranial gliomas, 255 were astrocytomas, and among 784 intracranial gliomas in our own material, 292 are astrocytomas.

Of the 5 spinal intramedullary astrocytomas in this series, 3 occur in the upper cervical spinal cord, 1 occupies the parenchyma from the upper cervical to the upper thoracic region, and 1 infiltrates the lower thoracic and upper lumbar segments. One of the 3 tumors in the upper cervical cord invades the subarachnoid space and extends upward into the leptomeninges about the brain stem, cerebellum, and medial surface of the right temporal lobe. There is a syringomyelia in the upper cervical segments which is slitlike and asymmetrically displaced to the left, but is more centrally situated as it extends in the cephalic direction and becomes continuous with an oval syringobulbia in the dorsal portion of the lower medulla.

The color of the tumors is grayish white, reddish gray, reddish blue or greenish blue. One is soft, and one moderately firm, and the latter contains many small cysts filled with glairy material. Cyst formation is relatively more common in intracranial astrocytomas, particularly those of the cerebellum. Three tumors are firm and tough.

Bailey and Cushing (1926) subdivided their astrocytomas into protoplasmic and fibrillary types. Since most astrocytomas are made up of both these cell types (and occasionally of variable numbers of oligodendroglia cells as well), this subdivision was dropped by Cushing (1932) and then by Bailey (1933). Penfield (1937) discarded this subclassification since he felt that there were no true protoplasmic astrocytes in tumors and substituted another: (1) pilocytic astrocytoma (made up of piloid astrocytes described below); (2) gemistocytic astrocytoma (composed of large-bodied, plump astrocytes—*gemästete glia Zellen* of Nissl and astrocyte with hyalinized body of Bailey, 1932); and (3) diffuse astrocytoma (a tumor pictured as a loose-meshed, fibrillar neoplasm in which the astrocytes appeared shrunken and neural elements were included—although this category was not clearly defined). The two astrocytomas which Penfield found among 20 intraspinal gliomas were classified as pilocytic. Kernohan (1932) retained the subdivisions, protoplasmic and fibrous (fibrillary), in his classification of spinal cord gliomas. [Bailey (1938) has recently reaffirmed his belief in the occurrence of these two types of astrocytoma which, as he points out, were also accepted by Hortega (1932).] He found 2 fibrous and 2 protoplasmic astrocytomas in the spinal cord and 3 fibrous and 2 protoplasmic extramedullary astrocytomas arising from the intradural filum terminale.

All 5 of our astrocytomas occur in the spinal cord, none having arisen from the filum terminale. Although the tumor, which was described as soft grossly, contains a preponderance of protoplasmic astrocytes, fibrillary astrocytes are also present. The moderately firm tumor has a slight preponderance of fibrillary astrocytes. The other 3 tumors are composed almost entirely of fibrillary astrocytes, although small numbers of protoplasmic astrocytes are encountered in them. In these 3 there are many large areas in which the piloid form of fibrillary astrocyte is present exclusively. In view of the mixture of astrocytic cell types in each neoplasm, it is proposed here to use the designation astrocytoma for all these tumors composed of astrocytes, and in the individual description of a given neoplasm, to indicate what type of cell and what architecture predominates.

Area in which protoplasmic astrocytes predominate. Although Penfield denies the occurrence of protoplasmic astrocytes in astrocytomas, most other investigators agree that they occur and in some tumors predominate. In our single soft astrocytoma, these form the majority of cells in many areas. The tumor is moderately cellular and relatively avascular. Its cells show no special architectural arrangement; usually they are well separated and in some instances they are attached by one process to a blood vessel. This process can be seen to broaden into a so-called sucker foot at its point of attachment (Fig. 155). The cells are stellate, polygonal, and sometimes roughly rounded or oval, and have finely granular, palely acidophilic cytoplasm. The cells vary in size; some are large, have abundant cytoplasm, and large radiating cytoplasmic processes (Fig. 155). In some areas these large cells have homogeneous, shining cytoplasm and appear somewhat swollen, but they are not numerous. [This appearance has been referred to by Bailey (1932) as astrocyte with hyalinized body (from the illustrations evidently a protoplasmic astrocyte), and would appear to account for at least part of the elements in Penfield's gemistocytic astrocytoma, although Penfield insisted that they were all fibrillary, since he believed that hypertrophy could not occur in an astrocyte without conversion to the fibrillary form.] The nuclei in these large cells are usually eccentric, and are oval and light-staining, containing a small amount of finely and

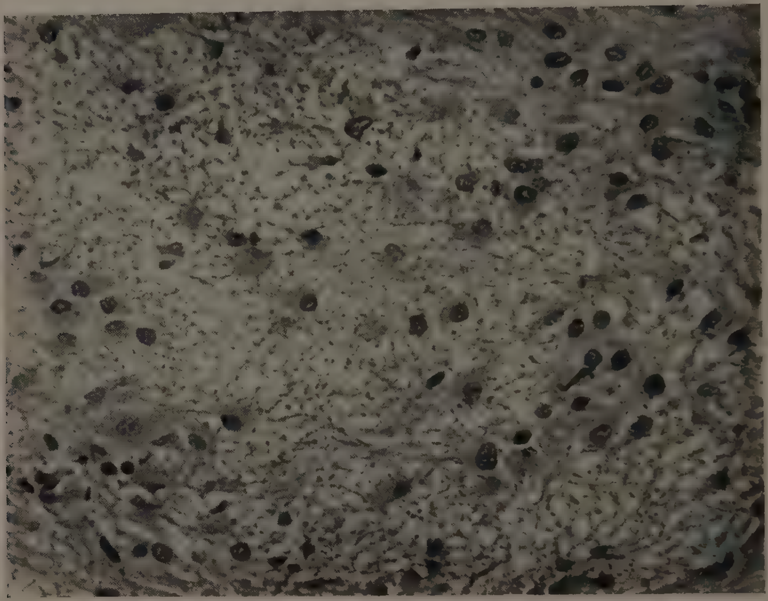


FIG. 154. Astrocytoma. The neoplasm was intramedullary from C 3 to T 4. The tumor is moderately cellular and its interstitial tissue is loose-meshed and fibrillar. The neoplastic elements have typical stellate bodies with homogeneous cytoplasm, numerous radiating fibrous processes and oval, comparatively light-staining eccentric nuclei. Phosphotungstic acid-hematoxylin stain. ×230.

coarsely granular chromatin (Fig. 155). Other smaller stellate or irregular cells with fewer processes and less cytoplasm have a similar type of nucleus, which is oval or spherical and usually centrally placed. The intercellular space is occupied by interlacing protoplasmic processes of the cells. Occasionally, a binucleated or multinucleated astrocyte which is somewhat larger than the others, is seen. These tumor cells are impregnated by Cajal's gold sublimate stain in which they take a reddish purple coloration. No neuroglial fibers (stained blue by Mallory's phosphotungstic acid hematoxylin stain) are present. The blood vessels in most areas show no abnormalities in their walls, although a rare capillary shows endothelial hyperplasia. Amitotic cell division occurs but mitoses are not seen. Such protoplasmic astrocytes are present in considerable numbers in the astrocytoma described grossly as moderately firm, although they do not predominate.

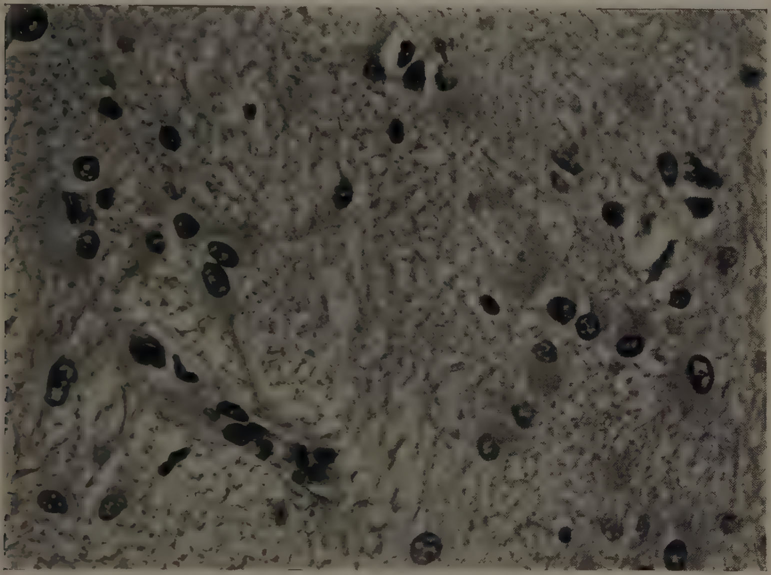


FIG. 155. Astrocytoma. The tumor was intramedullary from C 3 to T 4. The astrocytes are of varying size. Some are quite shrunken and their outlines are difficult to discern, the nucleus becoming prominent. Others are large-bodied, stellate and polygonal cells with homogeneous cytoplasm and eccentric, oval, light-staining nuclei. Note the process of one cell attached to a capillary wall. Hematoxylin eosin stain. ×460.

Area in which fibrillary astrocytes predominate. These zones appear fibrous and are usually sparsely cellular, although fairly cellular zones are encountered. The intercellular, fibrillar meshwork is loosely interlaced (Fig. 156) or densely woven. The cells are stellate, polygonal, irregular or elongated, and vary in size. Some are quite large and have abundant granular or homogeneous cytoplasm and many radiating fibrous processes (Figs. 154, 156). Some of the fibers are attached to blood vessels and broaden out into a sucker-foot at the point of attachment. More often, they are small, stellate, and have scanty cytoplasm and a moderate number of radiating fibrous processes. The nuclei of the larger cells are approximately like those of the protoplasmic astrocytes, while those of the smaller elements are usually hyperchromatic, smaller, at times spherical, and are central

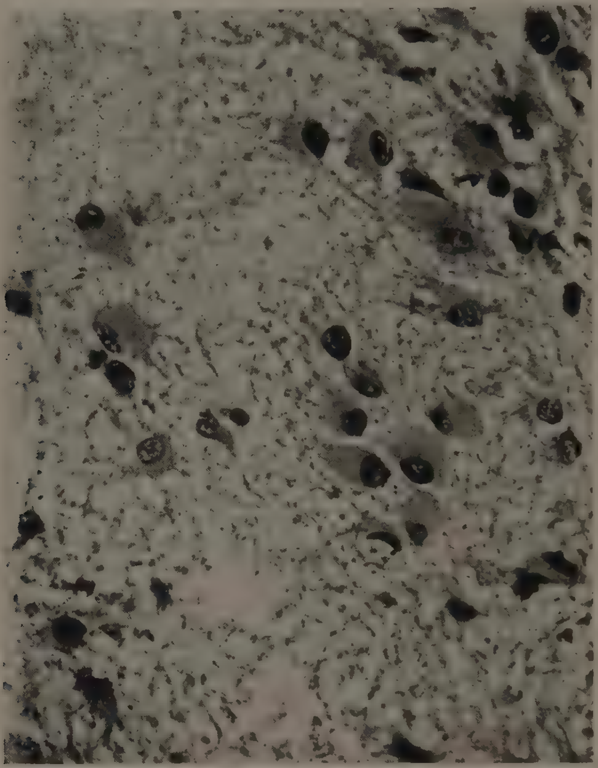


FIG. 156. Astrocytoma. The neoplasm was intramedullary from C 3 to T 4. The tumor is sparsely cellular and fibrillar. The tumor cells are large-bodied, stellate or polygonal and have abundant homogeneous cytoplasm and multiple fibrillary processes. The nuclei are oval and eccentric. Hematoxylin eosin stain. ×460.

(Figs. 154, 156). The predominating small cells, however, are the elongated bipolar elements with their fibrous processes extending from each extremity of the cell. They have scanty cytoplasm and a

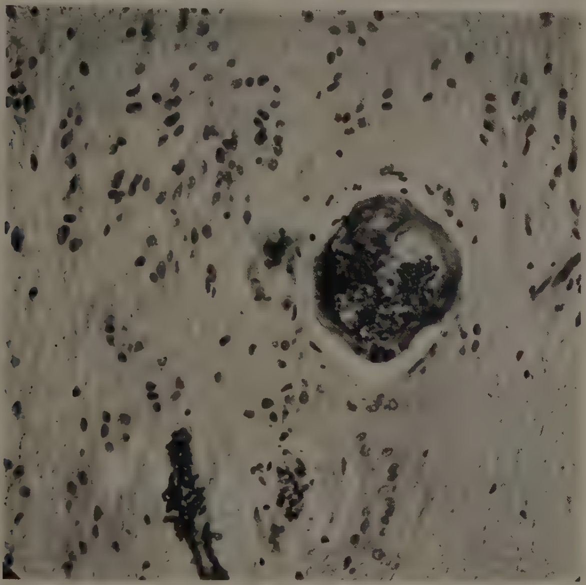


FIG. 157. Astrocytoma. The neoplasm was intramedullary from C 3 to T 4. The tumor tissue is sparsely cellular and richly fibrillar. Note the elongated bipolar astrocytes in places, so-called piloid astrocytes (Penfield). Hematoxylin eosin stain. ×300.

central oval, hyperchromatic nucleus (Fig. 157). These cells are often arranged in parallel rows with their processes streaming parallel to the long axis of the cell and, as has been pointed out by Kernohan (1932), they resemble the fibrillary astrocytes found in the white matter of the spinal cord, especially in tract degeneration. Penfield (1937) named this type of cell a piloid astrocyte because of the long slender fibers which form a feltwork in parallel sheaves (Gr. *pilos*: hair wrought into feltwork) (Fig. 157). Since these cells predominated in his two spinal cord astrocytomas, he referred to them as pilocytic astrocytomas. Kernohan's (1932) 5 intraspinal fibrillary astrocytomas seemed also to be composed of such elongated astrocytes. Occasionally, a fibrillary process of one of these cells can be seen attached to a vessel. The cells are impregnated deeply with Cajal's gold sublimate method and their processes are more slender, tougher-looking, and darker than the cytoplasmic processes of protoplasmic astrocytes. Often fine fibrils can be seen passing through the cell body from fiber to fiber. The fibers stain a deep blue in the phosphotungstic acid hematoxylin stain. Mitoses are not seen and amitotic division occurs infrequently. There are comparatively few blood vessels and these are mainly normal in appearance, although some precapillaries and arterioles may show fibrosis and hyalinization of their walls, and some capillaries, subendothelial fibrosis. In the astrocytoma described grossly as moderately firm, although there are areas in which protoplasmic astrocytes predominate, the fibrillary areas are more numerous and in these areas numerous small cysts occur. Among intracranial astrocytomas, cystic degeneration is more common in those that have been designated fibrillary; Penfield (1937) found cyst formation most common in his group of piloid astrocytomas. No calcium deposits are present in any of our 5 astrocytomas, nor does Kernohan describe any in his series. This is unlike the intracranial astrocytomas, in which calcification is relatively frequent.

In the 4 astrocytomas in which fibrillary astrocytes predominate, slightly in 1 case and markedly in 3, the piloid type of fibrillary astrocyte is the most common (Fig. 157). This is in accord with the findings of Penfield and of Kernohan.

Since the piloid astrocyte is the common type of reactive astrocyte in the tracts of the spinal cord, it is not surprising that some of the astrocytomas arising from the cord should be composed in great part of such cells. As the work of Kernohan and Harmier has shown, astrocytes occur in the conus medullaris and filum terminale, and are probably the cells of origin of the extramedullary astrocytomas of the cauda equina region. Kernohan (1932) mentioned another possible source for such tumors, namely, heterotopic masses of glial cells in the subarachnoid space of which he had observed five instances. In the group of 95 intraspinal gliomas of Rasmussen, Kernohan and Adson (1940), 6 extramedullary astrocytomas were listed.

Bailey (1938) called attention to the tendency to cellular polymorphism in some astrocytomas, particularly the cerebral variety, and felt that this ran parallel to the proneness of many cerebral astrocytomas to increase in malignancy. Scherer (1940), in an intensive study of 125 gliomas found that the majority of cerebral astrocytomas contained foci of malignant transformation (glioblastomatous). Kernohan and Parker (1932) described 4 gliomas of the spinal cord in a case of von Recklinghausen's disease, 1 of which was an astrocytoma, 1 a mixed astrocytoma and ependymoma, and 2 ependymomas. There was an associated syringomyelia. Four of the 8 gliomas of the spinal cord reported by Bailey and Foerster (1936) were astrocytomas, and of these, 3 were designated as protoplasmic.

Glioblastoma Multiforme

Glioblastoma multiforme (spongioblastoma multiforme, gliosarcoma, gliomes à cellules polymorphes, glioma sarcomatodes).

This malignant type of glioma was first separated from the welter of "gliosarcomas" by Strauss and Globus (1918), who named it spongioblastoma. Later, Roussy, Lhermitte and Cornil (1924) suggested the term glioma multiforme for the tumor, and Globus and Strauss (1925) modified their designation to spongioblastoma multiforme (describing one instance of its occurrence in the spinal cord). Bailey and Cushing (1926) accepted the latter designation, although they weighed the term glioblastoma multiforme. This last term was later agreed upon by many as more apt (Roussy and Oberling, 1931; Bailey, 1932; Cushing, 1932; Hortega, 1932, 1934; Bergstrand, 1932). The name spongioblastoma was to be reserved for those tumors made up more exclusively of spongioblasts. The new term implied a tumor made up of a variety of glial cell types, many of which are immature or anaplastic.

Only one of these malignant gliomas is present among our 26 intraspinal gliomas and this grew in the substance of the upper thoracic spinal cord. Of Penfield's (1937) 20 intraspinal gliomas, 3 were glioblastomas, and of the 95 intraspinal gliomas of Rasmussen, Kernohan and Adson (1940) 4 were of this type (designated as spongioblastoma multiforme) of which 1 was extramedullary. One of the 8 intraspinal gliomas of Bailey and Foerster (1936) was of this malignant variety. Again, as in the case of the astrocytoma, there is a marked difference from the intracranial percentage frequency. There are 250 glioblastomas among our 784 intracranial gliomas. This agrees with the findings in other large groups of intracranial gliomas. Glioblastomas stand second only to the intracranial astrocytomas in their frequency. In the spinal canal, however, they are relatively rare.

The one glioblastoma in our series was found within the upper thoracic spinal cord, swelling, distorting, and discoloring it grayish blue. On incision of the parenchyma, pinkish red, soft tumor tissue containing tiny yellowish punctate areas was extruded. Our larger glioblastomas of the cerebrum are soft and variegated in color, gray, yellow, brown, red and orange, due to necrosis and hemorrhages of different ages. Their margins are diffuse or moderately sharp, cream-colored, and granular. Often they contain small cysts which are filled with greenish or yellowish gelatinous fluid.

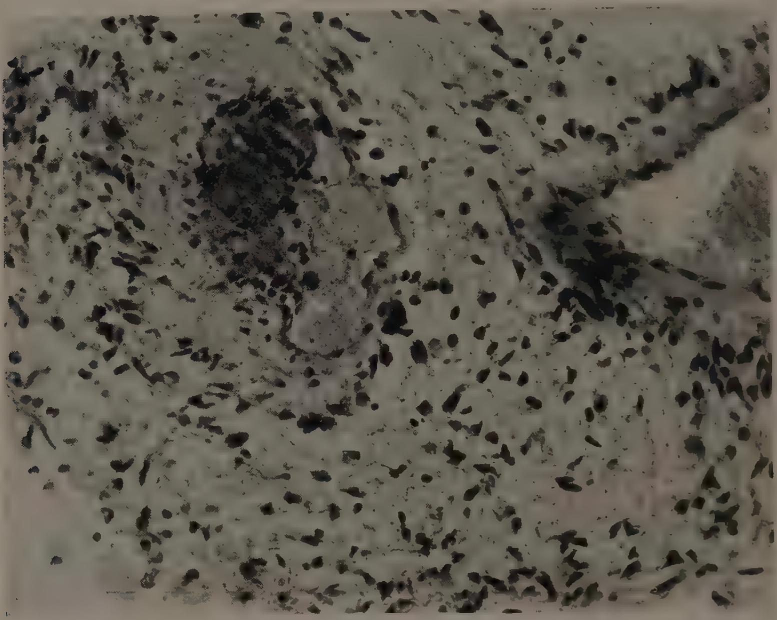


FIG. 158. Glioblastoma. The tumor was intramedullary from T 3 to T 5. The cells are variable in size, contour and in type of nucleus. A multinucleated giant cell can be seen adjacent to the vessel. The small artery is thrombosed. There is endothelial hyperplasia of the vessels. Hematoxylin eosin stain. ×230.

Our spinal cord glioblastoma is fairly cellular and varies from moderately to markedly vascular (Fig. 158). The cells show no special architectural arrangement except for occasional perivascular clustering. (Parallel arrangement of the bipolar elongated cells may be seen in some glioblastomas.) The tumor elements vary in size and shape. Some are elongated, unipolar and bipolar cells; others are pyriform, fusiform, spindle-shaped or bizarre and irregular in conformation (Figs. 158, 160). These cells have oval or elongated nuclei

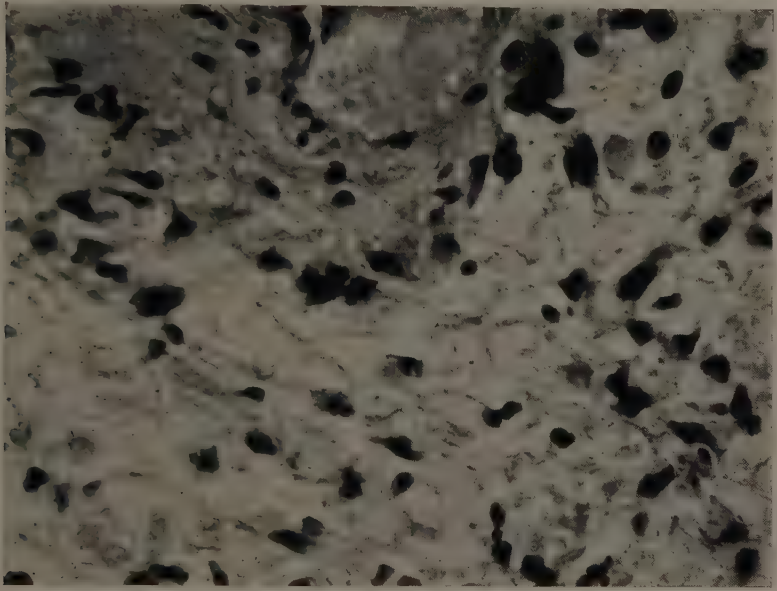


FIG. 159. Glioblastoma. The tumor was intramedullary from T 3 to T 5. Note the variability of the glial cell types and the multinucleated giant cell. Hematoxylin eosin stain. ×460.

and a few are attached to blood vessels at one end. Small cells with scanty cytoplasm, round or oat-shaped bodies, and hyperchromatic nuclei are also present (Fig. 159). Small numbers of large-bodied, round or stellate cells with homogeneous or granular cytoplasm, eccentric, light-staining, oval nuclei, and varying numbers of processes are seen (these processes are rarely fibrillary so that neuroglial fibers are absent or rare) (Fig. 160). Mitoses are moderate in number in this neoplasm, but are often very numerous in cerebral glioblastomas. The variability of cell types is attributed by Bailey (1938) to anaplastic variegation of cells with no close resemblance to embryonic elements. Globus and Strauss (1918, 1925), Bailey and Cushing (1926), Penfield (1937), and others, however, considered the unipolar and bipolar cells described above as spongioblasts. Small focal areas of necrosis are present in which the tumor cells are completely necrotic; these areas contain cellular debris, lipoid-laden mononuclear phagocytes, and occasional polymorphonuclear leucocytes and lymphocytes. No pseudopalisades are seen in this particular tumor. (This secondary heaping-up of tumor cells on either side of a columnar focal necrosis is a characteristic feature of cerebral glioblastomas, as are large diffuse areas of total necrosis.) Occasional large multinucleated cells with polygonal and irregular bodies and varying numbers of protoplasmic processes are seen (Figs. 159, 160). The capillaries show moderate endothelial hyperplasia (this is usually marked in cerebral glioblastomas), and a few arterioles show fibrosis, partial hyalinization, and thickening of their walls with narrowing of their lumina. A number of these are thrombosed and their walls are necrotic (Fig. 158). A few small arteries show adventitial hyperplasia. (Recent hemorrhages and hemosiderin-laden phagocytes are found in cerebral glioblastomas, but were not seen in this tumor.) Four of 5 intraspinal glioblastomas (spongioblastoma multiforme) described by Kernohan (1932) differed in some respects from the description just given, while 1 was similar to it. The cells were de-

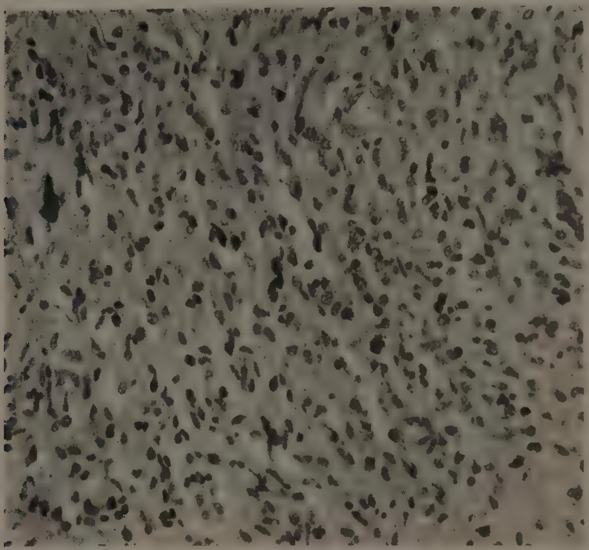


FIG. 160. Glioblastoma of spinal cord. The tumor is fairly cellular and includes a variety of neoplastic glial cell types. Note the elongated bipolar spongioblasts with their long oval nuclei, a variety of irregular cell forms, and multinucleated giant cells. Hematoxylin eosin stain. ×350. (Section by courtesy of Dr. Kernohan.)

scribed as varying in size, and having scanty processes and varying-sized nuclei (Fig. 160). Giant cells and giant nuclei were common. These were also frequent in the single glioblastoma recorded by Bailey and Foerster (1936). Multinucleated cells were frequent, as were mitotic figures. Degeneration of tumor tissue was uncommon and there were few if any reactive changes in the blood vessel walls. The neoplasm noted by Bailey and Foerster (1936) showed these features. Kernohan compared his glioblastomas to the so-called giant cell glioma of Stroebe. Penfield (1937) mentioned 3 intraspinal glioblastomas among 56 such tumors of the central nervous system, but did not describe them separately. Presumably, they were of the same type as his cerebral neoplasms.

OLIGODENDROGLIOMA

Oligodendroglioma (oligodendrocytoma, oligodendroblastoma). Like the astrocytoma and ependymoma, this is a glioma made up of adult glial cells, the oligodendroglia (Fig. 137). These cells were considered indifferent or immature elements (together with the microglia) until Hortega (1921) demonstrated their true nature as adult interstitial cells with his silver carbonate and other metallic impregnation methods. No example of this type of glioma occurred among our 26 intraspinal glial tumors.

There were 3 such neoplasms among the 95 intraspinal gliomas of Rasmussen, Kernohan and Adson and none among the 20 intraspinal gliomas of Elvidge, Penfield and Cone (1937), a fact which confirms its rarity. Oligodendrogliomas are uncommon in the intracranial cavity as well; only 36 of 784 gliomas in our material are of this type, a proportion which corresponds to that of other large series.

The 3 intraspinal oligodendroglial tumors of Rasmussen, Kernohan and Adson were all intramedullary. Kernohan (1932) reported one such tumor in the cauda equina region, presumably arising from the filum terminale.

The gross appearance of the intraspinal tumors was not stated in the reports. Their cerebral counterparts are usually well demarcated, grayish or grayish pink, and fleshy or granular and fairly firm. In some instances, particularly in children, they may be cystic.

Histologically, the oligodendroglioma (as seen in our cerebral specimens) is found to be fairly cellular and quite uniform in appearance due to its regular architecture and the uniformity of its cells (Fig. 161). These elements are small or of moderate size, sharply or indefinitely outlined, closely approximated, and are polygonal or irregularly rounded in shape. The close approximation and polygonal shape of the cells results in the appearance of a mosaic at times. The nuclei are spherical and hyperchromatic, containing a considerable amount of chromatin (Fig. 162). Mitoses are uncommon although occasionally they may be frequent. A striking and characteristic feature which frequently produces a weblike, lacy, honey-combed, or cribriform appearance of the tumor tissue is the feeble staining or lack of staining of a wide ring of cytoplasm about the nucleus, producing perinuclear halation (Fig. 162). These cells resemble ordinary oligodendroglia which have undergone swelling. Occasionally the cytoplasm of the cells is more distinct, the cell body small and oblong, short processes may be made out, and the spherical nucleus is eccentric. At times the cell bodies may be larger and the nuclei larger and more pallid. These cells have been considered younger oligodendroglial elements, oligodendroblasts. When a tumor has contained many such cells, it has been referred to as an oligodendroblastoma (Penfield, 1931; Kernohan, 1932; and others). Bailey and

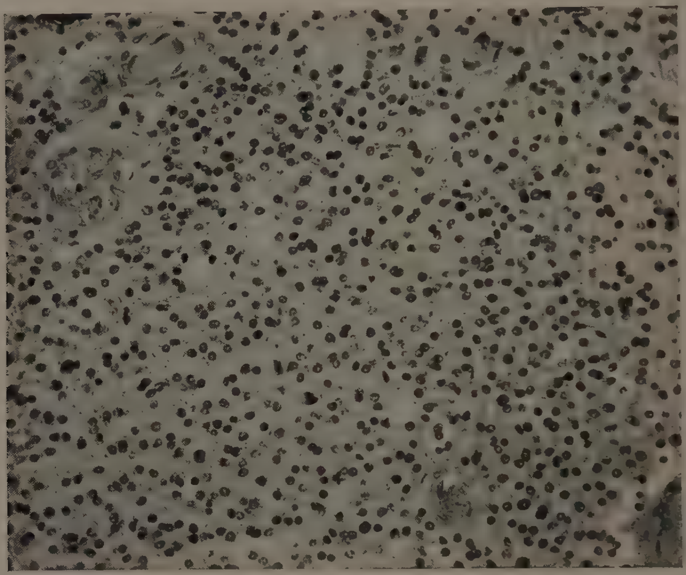


FIG. 161. Oligodendroglioma. The tumor is moderately cellular and uniform in appearance. The cell outlines are rounded or polygonal; the nuclei are spherical, hyperchromatic, and fairly uniform in size and shape. There are perinuclear halos. Hematoxylin eosin stain. ×230. (Section by courtesy of Dr. Kernohan.)

Bucy (1929) described mixed oligodendrogliomas and astrocytomas, and in such tumors and among such cells with more abundant cytoplasm, reported what they considered to be cells intermediate between oligodendroglia and astrocytes. Small numbers of astrocytes are often found in oligodendrogliomas. Blood vessels may be relatively infrequent (Fig. 161) or numerous; and, rarely, endothelial hyperplasia of capillaries and fibrosis and partial hyalinization and thickening of the walls of arterioles may be encountered. Calcium deposition is frequent and abundant in the cerebral oligodendrogliomas but has not occurred in the spinal examples. The cells of these tumors may at times be specifically stained by the silver carbonate method of Hortega, aiding in the demonstration of their oligodendroglial nature, but the attempt is rarely successful. Greenfield and Robertson (1933) reported a delicately staining, light blue reticulum among the cells in oligodendrogliomas stained by Mallory's connective tissue stain, which they consider characteristic for this tumor, and which we have seen in some of our oligodendrogliomas.

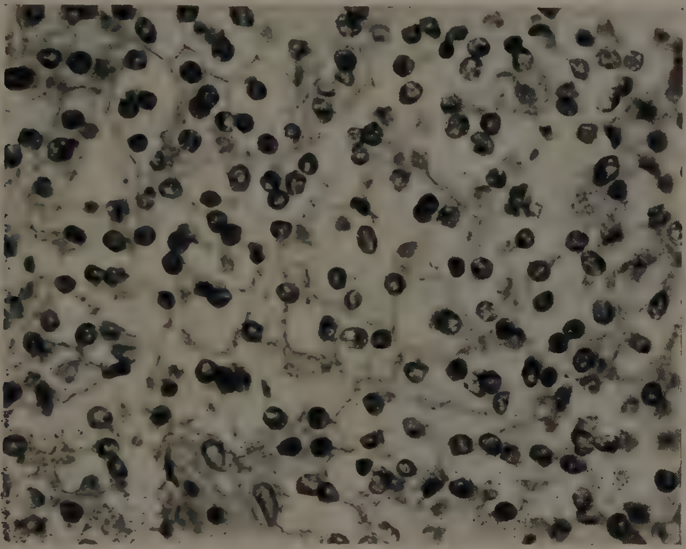


FIG. 162. Oligodendroglioma. The cell outlines are rounded or polygonal. The nuclei are roughly spherical, hyperchromatic and uniform. Note the perinuclear halos. Hematoxylin eosin stain. ×460. (Section by courtesy of Dr. Kernohan.)

As already noted, the Kernohans (1937) pointed out that oligodendroglial areas frequently occur in ependymomas. Of the 3 intraspinal oligodendrogliomas which Kernohan (1932) described earlier, the 2 intramedullary neoplasms contained areas resembling cellular ependymomas.

In other ways, they were like the cerebral oligodendrogliomas, as was the single extramedullary oligodendroglioma arising from the filum terminale, except that none contained any calcium.

The origin of these tumors involves exactly the same considerations as does that of the astrocytomas previously discussed, since oligodendroglia occur not only in the spinal cord proper but in the filum terminale as well (Harmier).

Astroblastoma

Astroblastoma (astrocytome pseudopapillaire). The astroblast is an immature glial cell derived from the primitive spongioblast by way of the bipolar and unipolar spongioblast, and is a direct precursor of the adult astrocyte (Fig. 137). It is polygonal, pyriform or roughly stellate in shape, has a moderate amount of cytoplasm, one heavy cytoplasmic process which is attached by a sucker-foot to a blood vessel, and varying numbers of short, immature processes arising from the opposite pole of the cell (Fig. 137). The nucleus is usually oval and light-staining. The cell is impregnated deeply with Cajal's gold sublimate method.

No example of a tumor composed of such cells was present among our 26 intraspinal gliomas. Kernohan (1932) described 2 intraspinal neoplasms of this type, 1 being intramedullary and 1 lying in the subarachnoid space. Kernohan, Woltman and Adson (1933) found 3 examples among their 25 glial tumors of the cauda equina region, and Elvidge, Penfield and Cone (1937) mentioned 2 in the same locus among their 20 intraspinal gliomas. Only 7 astroblastomas were present among our 784 intracranial gliomas while 35 were present in Cushing's 687. (This difference probably depends upon a disagreement in histological diagnosis rather than upon a varying incidence. In any case, these tumors are not common.)

The gross appearance of these intraspinal astroblastomas is not described, but their intracranial counterparts may be soft or moderately firm; have indefinite edges; be granular and grayish or, less often, show multicolored areas of degeneration like the glioblastoma; and on occasion they may be cystic.

Histologically, they appear loose-meshed and are quite vascular. Cells of the type just described as astroblasts are found radially arranged around blood vessels. The nuclei of these neoplastic cells vary considerably in size and shape, contain a good deal of fine and some coarsely granular chromatin, may be light or deep-staining, and are frequently eccentric. The cells are attached to the blood vessels by a prominent sucker-foot. Degeneration of tumor cells may be intense in the intervascular areas. Varying numbers of astrocytes and spongioblasts and occasional multinucleated giant cells may be present. Kernohan (1932) found multinucleated cells rare in his two intraspinal astroblastomas and considered this a point of difference from their intracranial counterparts. Mitoses are not frequent. It is obvious that this tumor is similar to the glioblastoma multiforme, and Bailey (1932) placed it between that tumor and the protoplasmic astrocytoma into either of which it may blend. Its recognition depends upon the observation of the predominance of astroblasts. The resemblance of this tumor to ependymoma and its differentiation from that neoplasm has been discussed before.

The origin of this tumor and of others of the tumors to be described, which are composed in large part of "immature" glial cells, involves the same considerations discussed under glioblastoma multiforme.

POLAR SPONGIOBLASTOMA

Bipolar, and in turn unipolar spongioblasts, are immature glial cells derived from primitive spongioblasts, and they are precursors in that order of the astroblasts which give rise to adult astrocytes (Fig. 137). They are elongated, unipolar or bipolar cells with oval or elongated, deeply staining nuclei, with the cytoplasm scanty about the nucleus, but often extending for long distances beyond its extremities as slender cytoplasmic processes, as can be best seen in a supravital preparation. One extremity may rarely be traced to a blood vessel, but without the formation of a sucker-foot. The cells do not form fibrils.

No tumors composed of such cells were present among the intraspinal gliomas of the present series. Kernohan (1932) described 2 intraspinal spongioblastomas, among 65 intraspinal gliomas, both of which were intramedullary. Elvidge, Penfield and Cone (1937) found 3 such tumors among their 20 intraspinal gliomas, 1 of which was intramedullary, while the other 2 occurred in the cauda equina region. One polar spongioblastoma occurred among the 8 intraspinal gliomas of Bailey and Foerster (1936). These again are rare intraspinal gliomas, nor are they common in the intracranial cavity. Among 784 intracranial gliomas in our material, only 27 were polar spongioblastomas, which agrees approximately with Cushing's (1932) findings of 32 such tumors among 687 gliomas.

The gross appearance of the few intraspinal spongioblastomas reported is not given. The equivalent tumor in the intracranial cavity is usually firm, grayish white or cream-colored, relatively avascular, somewhat fibrous in appearance, has indefinite margins, and is not infrequently cystic.

Histologically, the neoplasm resembles the nerve sheath tumor in many respects, which has led to the designation of some of these tumors as central neurinomas, as already noted (Josephy, 1924; MacPherson, 1925). Cells of the type described as unipolar and bipolar spongioblasts are arranged in parallel rows in columns which curve and interweave in many directions. The proportions of these two cell types vary, although the bipolar elements usually predominate. Their slender polar extremities streaming in bundles, and interlacing, produce much the effect seen in the nerve sheath tumor. They are often wavy and dense. Cyst formation may occur with amorphous material being present in the cysts, but necrosis of the kind seen in a glioblastoma is not usually encountered. Mitoses are rare, as are multinucleated cells, although according to Pilcher (1932) they appeared to be frequent in one series of such tumors. Calcification occurs in the cerebral variety. This neoplasm is distinguished from the nerve sheath tumor by the absence in it of true palisades (although suggestive nuclear clustering may occur), and, more importantly, by the absence of connective tissue fibers which are present in such abundance in the latter neoplasm. The spongioblasts should be impregnated by Cajal's gold sublimate method (often this is not easy to accomplish in this particular tumor).

Hortega (1932, 1934), supported by Roussy and Oberling (1932), advanced another opinion as to the nature of this tumor. He considered the tumor cell to be an elongated form of oligodendroglia and referred to the neoplasm as *oligodendrocytome, tipo fasciculado*, while Roussy and Oberling termed it *oligodendrocytome à cellules fusiformes*. Hortega considered oligodendroglia to be homologous with Schwann cells and so related these tumors to the nerve sheath tumors (neurinomas), accepting the Schwann cell theory of origin of the latter. Bailey (1938), who, with Eisenhardt (1932), described the tumors as spongioblastomas, contested Hortega's conclusions which were based on the specificity of Hortega's stain for oligodendroglia.

In addition to the suggestion that these tumors may be related to the nerve sheath tumors (neurinomas) noted above, or that they may be oligodendrogliomas, the question of the distinction between some of these tumors and astrocytomas composed of piloid astrocytes (piloid astrocytoma of Penfield) has been raised. Russell and Bland (1934), in preparing tissue cultures from gliomas, found that only astrocytes could be cultivated from their examples of polar spongioblastoma. They, therefore, indicated the possibility that many, perhaps most, of the polar spongioblastomas were really (piloid) astrocytomas. The objection may, however, be raised that since all cells do not grow out with equal readiness in tissue cultures, and some, as yet, not at all, polar spongioblasts were present in these tumors but did not wander out and multiply, while some associated astrocytes did. In any case, it is difficult to understand why this tumor (polar spongioblastoma), which is slowly growing and relatively benign, should be composed of such immature elements as spongioblasts.

MEDULLOBLASTOMA

Medulloblastoma (granuloblastoma, glioblastoma isomorphe, gliosarcoma, neurospongioma). Bailey and Cushing (1926) felt that it was necessary to accept Schaper's hypothesis of an undifferentiated bipotential immature neuroectodermal cell which could become either a spongioblast or a neuroblast, to explain certain features of gliomas, and they suggested the term medulloblast for such elements

(Fig. 137). They considered that a rapidly growing tumor of the cerebellum, occurring commonly in childhood, was composed of such very immature cells.

The cells of this tumor are small, have scanty cytoplasm, and are rounded or oat-shaped (Figs. 137, 168). The nuclei are spherical or oval and markedly hyperchromatic, uniform, and mitoses may be numerous or amitotic cell division may predominate (Fig. 168). The tumor is very cellular, relatively avascular, and the cells tend to cluster. In such clusters, the cells may be disposed in rows or arranged radially with the nuclei at the outer end of the cells outlining a circle or semicircle, and their bodies forming the very indistinct irregular spokes of a wheel (Fig. 168). Such formations are called pseudorosettes. The stroma is scanty when the tumor lies in the parenchyma, and is composed of small numbers of astrocytes and their fibers, and scattered blood vessels which appear relatively normal. Where the tumor so frequently invades the leptomeninges varying numbers of connective tissue fibers form the stroma.

Whether such bipotential cells occur during embryological development of the central nervous system has been doubted by His, who felt that cells of this type were either potentially spongioblastic or neuroblastic. Bailey (1938) has recently reaffirmed his belief that such migratory embryonic multipotential cells exist, and pointed to the embryological studies of Ostertag (1936) as confirmation. Ostertag demonstrated that what he considered to be such undifferentiated cells persist longest in the neighborhood of the nodulus of the cerebellum, and Bailey (1938) pointed out that this is the commonest site of origin of medulloblastomas. Bailey further referred to the closely related tumor of the adrenal, which Wright (1910) called neuroblastoma and which is now known as sympathogonioma, and to the fact that its constituent cells, the sympathogonioma, are bipotential in that they can give rise to neural elements and chromaffin cells. Bielschowsky (1932) stated that in some tumors, cells of both these lines may be found side by side. Stevenson and Echlin (1934) noted what they thought to be a persistence of the external granular layer of the cerebellum in some of their cases of medulloblastoma of that organ, and derived the tumor from the granular layers of the cerebellum. They suggested the term granuloblastoma for the neoplasm and classified its constituent cell as a neuroblast.

The term medulloblastoma has become widely accepted as serving to designate a very definite type of malignant glioma and should be retained.

No example of this tumor was present among our 26 intraspinal gliomas. Kernohan (1932) reported 4 intramedullary neoplasms of this type among his 65 intraspinal gliomas. Elvidge, Penfield and Cone (1937) found none among their 20 intraspinal gliomas. Rasmussen, Kernohan and Adson (1940) reported 3 intramedullary medulloblastomas among their 95 intraspinal gliomas.

Kernohan described his spinal intramedullary medulloblastomas as being typical. They were cellular and the cells were arranged in short rows or palisades with an occasional pseudorosette. The cytoplasm of the cells was scanty and their shapes as described above. In one tumor, many spongioblasts were present. Bailey and Cushing (1926), Bailey (1932), Elvidge, Penfield and Cone (1937), and others have described spongioblasts and neuroblasts [unipolar and bipolar cells having large oval, vesicular nuclei often with prominent nucleoli which stain specifically with silver stains for nerve cells (Fig. 137)] in such tumors of the cerebellum.

In a personal communication, Kernohan recently expressed some doubt as to these intraspinal neoplasms being true medulloblastomas, and others have been sceptical of the probability that such tumors arise elsewhere than in the cerebellum. Zimmerman (1940), however, reported the experimental production of cerebral medulloblastomas in mice by the inoculation of methylcholanthrene. Eden (1938) referred to an unpublished case of Foerster's to be included in a forthcoming book on tumors of the brain and spinal cord. This was said to be a medulloblastoma of the lumbar cord which spread widely in the subarachnoid space.

GANGLIONEUROMA

Ganglioneuroma (ganglioglioma, ganglioneuroblastoma, ganglioglioneuroma, neuroglioma, ganglionic glioma, etc.). This tumor is composed of ganglion cells and their unmyelinated or myelinated axons, and varying numbers of glial cells (neurilemmal cells in the peripheral nervous system), most often astrocytes. Varying numbers of what may be immature neural elements and perhaps also immature glial cells (neuroblasts and spongioblasts) may also be present. Although a variety of names have been suggested to designate the various combinations of mature and immature ganglion and glial cells present in this type of tumor, it would seem best to retain the designation commonly used for the similar tumor of the peripheral nervous system, ganglioneuroma, since it is widely accepted, and to describe individual variations in these neoplasms without further subclassification or complication of nomenclature.

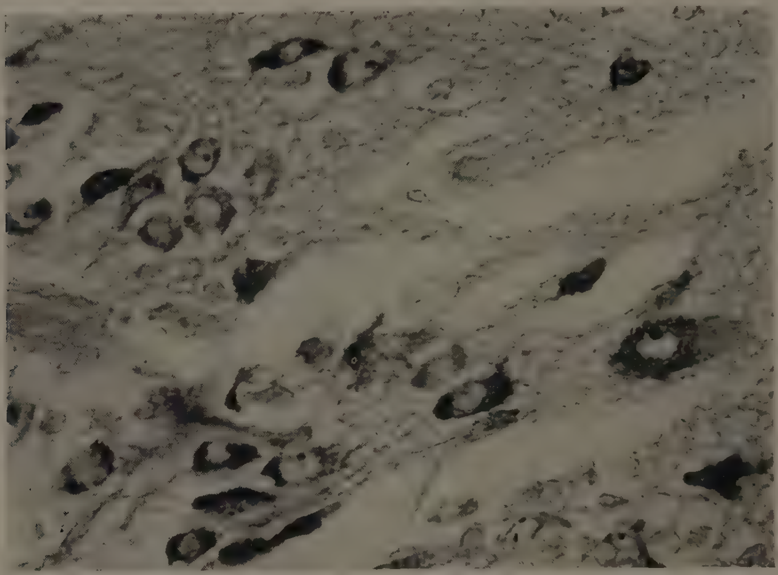


FIG. 163. Ganglioneuroma. The nerve cells are of varying size and shape, showing no constant orientation of their apical dendrites. All contain Nissl substance and have typical large, very light-staining nuclei and prominent nucleoli. Nissl stain. ×600. (Section by courtesy of Dr. Kernohan.)

This tumor, although rare in general, occurs most frequently in neural tissue or its derivatives outside of the central nervous system, in the sympathetic chain, adrenal medulla, and, less frequently, in the cranial and spinal nerves and ganglia. There are only 5 instances, all intracerebral, among our 784 intracranial gliomas, and none among our 26 intraspinal gliomas.

Among Cushing's (1932) 687 classified intracranial gliomas, 3 were of this type. Penfield (1937) reported no intraspinal or intracranial examples among 210 gliomas. Kernohan, Woltman and Adson (1931) noted 1 ganglioneuroma among 42 intraspinal gliomas, Kernohan (1932) 2 among 65, and Rasmussen, Kernohan and Adson (1940) among 95 intraspinal gliomas found 2 intraspinal intramedullary ganglion cell tumors and 2 extramedullary ganglioneuromas. Foerster (1924) and more recently Lichtenstein and Zeitlin (1937) described single examples of this tumor confined to the spinal cord. Pick and Bielschowsky (1911) and Foerster and Gagel (1932) recorded ganglioneuromas of the brain stem which secondarily invaded the first few segments of the cervical spinal cord.

The cerebral ganglioneuromas are usually firm, grayish, grayish white or grayish red, lobulated or nodular, sharply demarcated, and occasionally cystic. Kernohan described his intramedullary spinal ganglioneuromas as soft and purplish, or maroon-colored and encapsulated; and his extramedullary (sacral) as firm, almost fibrous, and encapsulated. Lichtenstein and Zeitlin's intramedullary spinal ganglioneuroma (ganglioglioneuroma) apparently extended through many segments of the spinal cord. It was light yellowish brown and soft in some areas, and grayish white and firm in others, occupying from one-third to almost two-thirds of the cross section of the spinal cord at various levels. A cavity or cyst of considerable size and varying form, usually central, extended throughout most of the tumor and was always surrounded by tumor tissue.

In our cerebral ganglioneuromas, the neoplastic nerve cells show a tendency to cluster or to be strung out in irregular formations without any architectural regularity. They show great irregularity in orientation and some variation in size. They contain Nissl bodies which are often deficient in numbers, irregularly distributed, and vary in size (often finely granular or dustlike) or are entirely devoid of tigroid bodies. Neurofibrils are present, most often detectable in the dendrites. The nuclei are large, vesicular, pale, and contain prominent nucleoli. Binucleated and multinucleated nerve cells are encountered. In the ganglion cell clusters, smaller cells are also present in varying numbers. Some are elongated, pyriform, unipolar or bipolar elements with nuclei similar to those of the adult cells, and contain occasional Nissl granules. Others are small, rounded or oat-shaped, and have scanty cytoplasm and spherical hyperchromatic nuclei. At the margins of the ganglion cell clusters and mingled with them are fibrillary astrocytes, many of which are shrunken or elongated and bipolar, the piloid variety. Some of the bipolar cells may be spongioblasts. Bailey (1932) and others believed that these elongated elements are related to the Schwann cells and capsular cells of the peripheral nervous system, such as are seen in peripheral ganglioneuromas. Mitoses are not present, cell division being apparently amitotic. Blood vessels are moderately abundant and are chiefly capillaries. In places there is a subendothelial increase in connective tissue fibers of the capillaries, hyalinization of the walls of the arterioles, and thickening of the adventitia of large vessels. In one of our cerebral ganglioneuromas there is considerable fibrosis of vascular origin with the production of numerous connective tissue trabeculae of varying width, which traverse the tumor and often surround islands of ganglion and glial cells producing a lobulated appearance (Wolf and Morton, 1937).

Kernohan (1932), in describing two spinal cord ganglioneuromas, reported that one was composed of almost mature uniform ganglion cells with typical nerve cell nuclei, finely granular Nissl bodies, and three or more dendrites but no axons (Fig. 163). Binucleated and multinucleated nerve cells were uncommon in contrast to the findings in ganglioneuromas elsewhere. A second tumor contained no mature forms but did contain bipolar, unipolar, and multipolar elements in equal numbers (a neoplasm which might be classed by some as a neuroblastoma or ganglioneuroblastoma, because of these immature elements).

The 2 spinal extramedullary ganglioneuromas of Rasmussen, Kernohan and Adson (1940), although so listed, were not intraspinal; they belong to that group of tumors occurring in the hollow of the sacrum, to which 3 of the extramedullary ependymomas described by the Kernohans and previously mentioned belonged—a group that was separately described by Adson, Moersch and Kernohan (1939). One of these tumors occurred in the bones of the sacrum, extended into the hollow of the sacrum, and also protruded posterior to it. The other arose from the hollow of the sacrum and almost filled the pelvis. Their gross appearance has been referred to previously. Histologically, they were almost identical and were typical ganglioneuromas. They contained numerous large, multipolar nerve cells, with large vesicular nuclei having prominent nucleoli. Nissl granules were numerous and intracellular neurofibrils, branching dendrites and axons were present. Although resembling sympathetic nerve cells, no endocapsular cells or ectocapsular cells were present in relation to them. Some showed large cytoplasmic vacuoles. This, the authors considered to be common in ganglioneuromas, especially in those arising from the sympathetic nervous system. The nerve cells occurred in clusters of 15 to 20 and multinucleated forms were fairly common. Both tumors had a rich connective tissue stroma through which bundles of unmyelinated nerve fibers and single fibers ran.

Foerster (1924) reported an intramedullary ganglioneuroma of the fourth to sixth cervical segments which was reddish and soft at the surface and grayish or brown, and firm or yielding on section. Only a narrow lunate band of cord parenchyma in this region remained uninvaded. In addition to mature ganglion cells, some of which were multinucleated, Foerster described many younger elements which he considered to be their precursors, particularly lymphocyte-like elements (apolar neuroblasts, medulloblasts). There were large areas whose architecture resembled the structure of nerve sheath tumors. The profusion of nerve fibers present in these zones, however, would seem to differentiate them completely from the nerve sheath tumors already described, as do other considerations (Wolf and Morton, 1937).

The ganglioneuroma ("ganglioglioneuroma") of Lichtenstein and Zeitlin (1937) contained many ganglion cells in pairs and nests scattered among dense masses of glial elements and numerous blood vessels. They possessed typical nerve cell nuclei, dustlike Nissl substance, neurofibrils, and occasionally were binucleated. Nerve fibers, both stout fibers and fine fibrils were abundant, and appeared to be far in excess of the nerve cells. They could at times be traced to a neoplastic ganglion cell. Glial cells and fibers (presumably astrocytes) were numerous, and nearest the large cavity or cyst which traversed most of the neoplasm, parallel longitudinal rows of elongated glial cells (not further characterized) were present, producing what the authors considered a neurinoma-like appearance (a resemblance which has been noted by many others—Schmincke, 1910; Greenfield, 1919; Olivecrona, 1919; Watjen, 1930; and others). Ganglion cells were absent or infrequent at some levels. Foci of old and recent hemorrhage and focal necroses occurred in the neoplasm and were evidently the basis of the cavitation and cyst formation to which the authors, it seems unnecessarily, applied the name, pseudosyringomyelia.

Friedman and Scheinker (1934) reported a ganglioneuroma of the cauda equina which for reasons stated by Wolf and Morton (1937) would appear to be an ependymoma.

The fact that adult ganglion cells are not known to divide, gives the embryonal rest theory some importance in relation to the origin of ganglioneuromas. Pick and Bielschowsky (1911) believed that every tumor of neural elements harks back to an abnormal deposition of multipotential embryonal nerve cells, due to a developmental defect in the embryo. In tuberculosis, which partakes of the characteristics both of malformation and neoplasia, neural and glial elements proliferate in compact foci. It is of interest that in the first group of 13 experimental gliomas induced in the brain of mice (Seligman and Shear, 1939) none was composed of neural elements. [The so-called experimental gliomas of Marburg (1937) and Weil (1938) have been criticized as being reactive gliosis rather than neoplasms.]

Bailey and Foerster (1936) referred to the frequent occurrence of spinal cord gliomas in the inner portions of the posterior columns, and their repeated association with columnar gliosis and syringomyelia in the same region. They discussed the probability of a causative relationship between these gliomas and some congenital abnormality in the formation of the posterior septum of the spinal cord, a hypothesis previously formulated by Ostertag (1925) and others. To apply such a theory of the origin of tumors to the majority of the gliomas of the spinal cord, however, would be to assign them a separate position among the gliomas of the central nervous system, a division which seems unwarranted.

Miscellaneous Gliomas

Neuroepithelioma. This tumor occurs only rarely in the central nervous system and did not appear in the series of the various spinal cord gliomas of Kernohan et al., nor of Penfield. None was found among the 26 intraspinal gliomas of the present series. They are typical tumors of the retina (said to be derived from the neuroepithelium of Schwalbe) where they are composed of rosettes of cells. The cell bodies are elongated and the cytoplasm is abundant and granular. The nuclei are oval and hyperchromatic, containing a good deal of chromatin. The cells are arranged radially around a cavity forming a rosette. The free borders may bulge into the lumen and be ciliated. Beneath the cilia is a row of blepharoplasten. The distal extremities of the cells terminate in an end bulb, on a vessel or at a connective tissue septum, an abundant network of connective tissue being present. The tumors of the central nervous system, which have been described as neuroepitheliomas, have had most of these characteristics. These tumors are considered to be composed of primitive spongioblasts (Fig. 137) which characteristically tend to arrange themselves about small cavities.

Rosenthal (1898), Bittorf (1904), and Marburg (1921) were considered by Bailey and Cushing to have described real neuroepitheliomas of the spinal cord although later Bailey and Foerster (1936) referred only to the case of Hartwell and Stevenson (1925) as an instance of this type of neoplasm. The tumor of Rosenthal was associated with a syringomyelia. Bailey (1938) referred to an unreported spinal cord tumor that he had seen, which might possibly be of this type, but he did not further describe it. Antoni (1936) quoted the cases of Kling (1907), Thielin (1908), and Riedel (1919) as being similar to Rosenthal's, and considered these tumors, as well as Bittorf's, to be ependymal gliomas. In the present discussion of intraspinal ependymomas, the difficulty of distinguishing between these tumors and some ependymomas was noted. It would seem that Kernohan included intraspinal tumors of this type among his ependymomas, and the benign nature of most of these neoplasms would seem to justify this classification. Foerster and Bailey (1936) and Bailey (1938) agreed in part, but Bailey felt that on the basis of malignancy, a category of tumors composed of primitive spongioblasts should be retained, neuroepithelioma, one of which he believed might be the unreported spinal cord tumor just referred to. In any case he noted that neuroepitheliomas are very rare and "play a small role in the brain and cord."

Neuroblastoma. The tumor which Wright (1910) originally called neuroblastoma of the adrenal is now known as a sympathogonioma. It is closely related to the medulloblastoma of the central nervous system. The term neuroblastoma was later reserved by Bailey and Cushing (1926) for tumors composed of unipolar neuroblasts (Fig. 137), and they pointed out that such tumors were exceedingly rare. These tumors were described as sharply demarcated, benign, quite cellular, fairly vascular, and composed of unipolar cells whose processes were unmyelinated. Bailey (1938) became skeptical of this category, for in reviewing the three tumors originally so classified by Cushing and himself, he reclassified one as an ependymoma and left the other two unclassified.

Kernohan (1938) described the cells of a neuroblastoma as having very little demonstrable cytoplasm, and being characterized by a large vesicular nucleus and a large prominent nucleolus. Mitoses were present and the tumor grew rapidly. In the more slowly growing type, Nissl substance could occasionally be seen in the cytoplasm and the cells had an affinity for silver in the Orlandi and Bielschowsky stains.

No such tumors were present in Penfield's series of 20 and in the present group of 26 intraspinal gliomas. Among their 95 intraspinal gliomas, Rasmussen, Kernohan and Adson (1940) reported 2 intramedullary tumors under the heading ganglioneuroma and neuroblastoma and these presumably were a mixture of mature and immature neural elements.

Medulloepithelioma. This was described by Bailey and Cushing (1926) as the most primitive of their tumors of neuroectodermal origin, reproducing some of the features of the medullary band and tube (Fig. 137). It is a highly cellular tumor, showing numerous mitoses. The cells tend to grow in long bands with limiting membranes on each side through which the cells break in places to form irregular masses. The cells are columnar and have large oval nuclei. Rosettes like those in the neuroepithelioma are often present, and it has been suggested that the medulloepitheliomas be included in that category.

Greenfield reported (1934) such a tumor in the sacral canal and believed that it arose from the filum terminale. The tumor extended upward, filling the lumbar sac, and invading the subarachnoid space in which it became disseminated as high as the base of the frontal lobes. Roussy and Oberling (1931), who included medulloepitheliomas among neuroepitheliomas, described a tumor in the sacrococcygeal spinal canal as *neuroépithéliome*, and Greenfield considered this neoplasm similar to his own. Both tumors were evidently slow-growing; Greenfield's patient had symptoms for six years. This would not accord with Bailey and Cushing's impression of the great malignancy of this type of neoplasm, nor with the suggestion that there is, in general, an inverse parallelism between the malignancy and the degree of differentiation of the chief cell type in gliomas. Greenfield agreed with the French authors and with Davie (1932) in explaining this slow growth of his medulloepitheliomas, by classing them as dysembryomas, rather than as "true cancers of the nervous tissue," as are medulloblastomas. At least in the instance of Roussy and Oberling's *neuroépithéliome*, the description and illustrations make one wonder whether these authors were not dealing with a tumor which Kernohan (1931, etc.) classed with the ependymomas and which Bailey (1938) is now rather inclined to accept as such. This alternative interpretation would be in harmony with the benign growth of most ependymomas and their common occurrence in the lower portion of the spinal canal.

No intraspinal medulloepithelioma is present in our series nor in the other series of intraspinal tumors to which reference has been made.

Extramedullary Gliomas

Those tumors in our series which occurred in the region of the cauda equina and probably originated from the filum terminale, have been described and discussed under the various glioma subdivisions as they occurred.

Among Rasmussen, Kernohan and Adson's (1940) group of 95 intraspinal gliomas, 9 gliomas were listed as extramedullary, 6 as astrocytomas, 1 as a glioblastoma (spongioblastoma multiforme), and 2 as ganglioneuromas. These were in addition to 32 ependymomas of the cauda equina region. Forty-one of their 95 intraspinal gliomas, then, were extramedullary. It is not clear how many of these were thought to have their origin in the subarachnoid space about the spinal cord, but it is clear that the great majority were in the cauda equina region and probably arose from the filum terminale. In an earlier report, Kernohan (1932) separated these two groups in a series of 65 intraspinal gliomas. Fifteen arose in the cauda equina region probably from the filum terminale, and 3 were described as possibly arising in the subarachnoid space about the spinal cord (cases previously referred to by Kernohan, Woltman and Adson, 1931). It was pointed out that these were biopsy specimens so that one could not be certain of their origin. The possibility that a pedicle attaching the tumor to the spinal cord had been overlooked in its surgical removal or that such a pre-existing pedicle had become atrophied, was discussed. A third hypothesis was postulated, namely, that the gliomas had arisen from glial heterotopias in the spinal subarachnoid space, first described by Wolbach (1907). Kernohan referred to the personal observation of five instances of such glial heterotopias containing various types of well differentiated glial or ependymal cells. Kernohan's gliomas of this group included astroblastoma and ependymoma. Orville T. Bailey (1936) described an astrocytoma of the cerebral leptomeninges, apparently with no point of origin in the cerebrum, which he derived from glial nests observed in the subarachnoid space in this case. He pointed out that such glial heterotopias seem to occur more frequently in the spinal subarachnoid space. Wolbach's (1907) original description of these glial nests concerned examples that occurred in that portion of the subarachnoid space. There were other associated congenital abnormalities in Wolbach's case, but these are not necessarily present in such cases. Thompson (1929) reported a spinal leptomeningeal astrocytoma as one of three "leptomeningiomas" of the spinal cord. It was the first case of the three, and was composed of large piloid or polygonal cells lying in a heavy meshwork of fibers which stained blue by the phosphotungstic acid-hematoxylin stain. Many of the spinal extramedullary gliomas reported in the French literature, on the other hand, were nerve sheath tumors, considered by their authors to be composed of Schwann cells and, therefore, designated as Schwannomas or peripheral gliomas. The neoplasm which was reported as a leptomeningeal glioma by Roussy, Cornil and Leroux (1923) was evidently a meningioma.

The possibility that an inapparent glioma of the parenchyma, spinal or intracranial, was present in all of these instances cannot be ruled out.

No "primary" gliomas of the spinal subarachnoid space occur in the present series nor does Penfield (1937) refer to any among his intraspinal gliomas.

Invasion of the Spinal Subarachnoid Space by Primary Gliomas of the Spinal Cord

Primary gliomas of the spinal cord do not commonly invade the leptomeninges. Two instances of such an invasion are present in our material. Its relative infrequency is curious when one considers that such tumors growing in an organ of limited dimensions commonly reach the surface of the spinal cord.

Older reports of such invasion are difficult to interpret, as for instance, the remarkable instance of Fischer's (1901) in which a "glioma sarcomatodes" of the spinal cord was said to have invaded not only the spinal leptomeninges, but the dura and vertebrae as well. Pels-Leusden (1898) reported a spinal cord glioma ("gliosarcoma") which invaded the spinal subarachnoid space and reached the cerebral subarachnoid space, but Eden (1938) voiced the justifiable suspicion that a mass referred to in the left lateral ventricle may have been the primary tumor. Grund (1906) described an intramedullary glioma which invaded the overlying leptomeninges. Rutimeyer (1930) reported a spinal cord glioma occurring in the lower portion of the spinal cord which invaded the subarachnoid space and spread up to the cervical region. Kernohan (1932) found only one instance of such subarachnoid invasion among his 65 intraspinal gliomas.

In this instance, the spinal cord had been split for decompressive purposes in the presence of an intramedullary oligodendroglioma. At a second operation, the tumor was found to have gained entrance to the subarachnoid space and spread for a short distance in the cephalad and caudad directions. There was a marked reactive fibrosis in the invaded leptomeninges, which will be referred to again in the discussion of secondary implantations in the spinal subarachnoid space of intracranial gliomas. Greenfield's (1934) medulloepithelioma of the sacral canal already referred to became seeded out in the subarachnoid space and reached the leptomeninges at the base of the frontal lobes. Eden (1938) reported an unclassified glioma of the spinal cord (glioblastoma?) which diffusely invaded the spinal subarachnoid space. Eden also referred to an unpublished case of Foerster of a medulloblastoma of the lumbar cord which spread throughout the subarachnoid space of the spinal cord and up to the cerebral leptomeninges.

One astrocytoma and one ependymoma in the present series of 26 intraspinal gliomas are widely disseminated in the subarachnoid space. The astrocytoma described previously as a high cervical intramedullary neoplasm invades the subarachnoid space and extends upward into the leptomeninges about the brain stem, cerebellum, and medial surface of the right temporal lobe. This is disseminated in the cephalad direction, as was the glioma reported by Greenfield already referred to. The tumor in the subarachnoid space is composed of the same type of elongated, piloid astrocyte present in the primary cervical neoplasm and contains a great many glial fibers and occasional multinucleated cells. There is considerable hyperplasia and fibrosis of the invaded leptomeninges and many of the leptomeningeal vessels show thickening and fibrosis of their walls. The ependymoma is the one described before as probably arising from the filum terminale, filling the lumbar sac, and spreading upward through the subarachnoid space (Fig. 138). Subarachnoid implants are found as high as the third thoracic cord segment. In the lumbar region, the tumor has rotated the cord through an angle of almost 90 degrees (Fig. 139). This rotation continues, becoming less marked, up to the third thoracic segment (Fig. 139). Histologically, the tumor in the subarachnoid space is very similar to the primary tumor in the cauda equina region. There is considerable hyperplasia and fibrosis of the leptomeninges in the invaded areas. In these zones, there is also marked obliterative endarteritis (Fig. 153), and calcification of leptomeningeal arteries, as well as calcification of thickened segments of pia and arachnoid membrane.

Secondary Implantations of Intracranial Gliomas in the Spinal Subarachnoid Space

The literature contains many instances of so-called primary sarcomatosis of the cerebrospinal leptomeninges, since the first description by Ollivier (1837). Mahon (1936) gathered 92 reports of diffuse tumor of the leptomeninges classified as either sarcomatosis or gliomatosis, the former predominating markedly. In analyzing the cases, he found that only 9 of the tumors could be accepted as true sarcomatosis; all the others were evidently instances of gliomatosis. The histological descriptions in the older literature are, however, for the most part too inadequate to permit reclassification in modern terms, so that the frequency with which various types of glioma invade the spinal subarachnoid space is not apparent.

Spiller (1907) reported a fourth ventricle ependymoma which spread down the subarachnoid space to the sixth cervical segment. A subarachnoid implantation was also present over the lower thoracic portion of the spinal cord. Schupfer (1908) recorded a "gliosarcoma" of the right temporal lobe which metastasized to the spinal leptomeninges with secondary invasion of the spinal cord. Gordinier and Sawyer (1911) described an ependymoma of the lateral ventricles and frontal lobes which became secondarily implanted in the spinal subarachnoid space. Lahmeyer (1913) reported a "glioma sarcomatodes" (glioblastoma?) of the cerebrum with secondary nodules in the leptomeninges of the spinal cord and cauda equina. Jacob (1916) recorded a cerebellar glioma (ependymoma?—Nelson, 1938) which not only became implanted in the walls of the lateral ventricles and cerebral leptomeninges, but in the spinal leptomeninges as well. Löwenburg's (1921) glioma (glioblastoma?) of the thalamus invaded the leptomeninges at the base of the cerebrum, and metastasized to the cerebellar and spinal leptomeninges. Bailey and Cushing (1925) in their description of cerebellar medulloblastoma pointed out the frequency with which this malignant glioma invades the subarachnoid space. Brannan (1926) reported another thalamic glioma (glioblastoma?) which acted as did Löwenburg's. Töppisch (1926) described a choroid papilloma which gave rise to numerous implants in the cerebral, cerebellar, and spinal subarachnoid space. The spinal implants extended from the midthoracic region to the cauda equina and were multiple and often confluent. Although Davis and Cushing (1925) have insisted that of the papillomatous tumors of the fourth ventricle, only the ependymoma may give rise to such secondary deposits, while the true choroid papilloma remains single, Cairns and Russell (1931) found evidence in one of their own cases that such a seeding may occur in an authentic choroid papilloma (this did not, however, result in spinal subarachnoid implants). Russell and Cairns (1930) described an astrocytoma of the thalamus with implants in the spinal leptomeninges. Bailey and Bucy (1931) suggested that the glial nodules in the leptomeninges in this instance were glial heterotopias, such as have been referred to previously (an opinion concurred in by O. T. Bailey in 1936); but Russell and Cairns (1931) in describing a series of cerebral gliomas with spinal subarachnoid metastases included this case, recapitulated their evidence, and reiterated their opinion that the leptomeningeal nodules were secondary implantations. These authors found that spinal metastases occurred in 8 of 22 gliomas of the brain in which both brain and spinal cord had been examined. They included the above cerebral astrocytoma, a cerebral glioblastoma and an ependymoma, three cerebellar medulloblastomas, an unclassified cerebral glioma and a retinal neuroepithelioma which had invaded the optic chiasm and cerebral leptomeninges from whence it had apparently spread to the spinal subarachnoid space. Ostertag (1934) stated that he had found intracranial and intraspinal metastases in 18 per cent of one year's series of brain tumors. Mittelbach (1935) reported a diencephalic astrocytoma which invaded the cerebral leptomeninges and became implanted in the spinal leptomeninges as well. Friedman and Plaut (1935) recorded a remarkable instance of dissemination of a pinealoma (pinealocytoma).

In the present series of 253 intraspinal neoplasms, there are 11 instances of dissemination of intracranial gliomas in the spinal subarachnoid space. These were encountered during a period when 784 cerebral gliomas were observed. It must be said at once that this wide divergence in findings from the frequency with which such spinal implants were seen in the series of Russell and Cairns (1931) and reported by Ostertag (1934) may be dependent in part upon the low incidence of spinal cord examinations in instances of cerebral

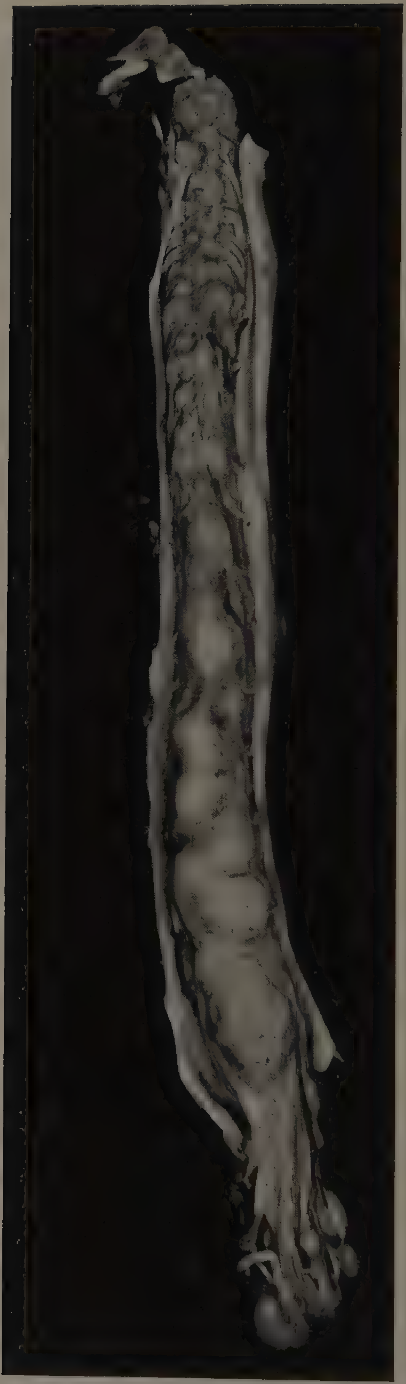


FIG. 164. Note the nodular and diffuse subarachnoid implantations of a medulloblastoma of the cerebellum. Nodules of the tumor are attached to the cauda equina.

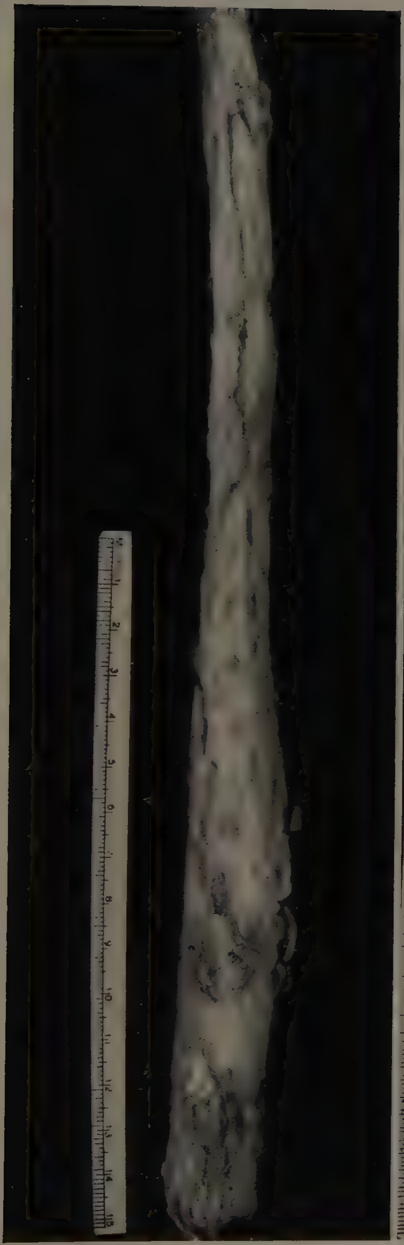


FIG. 165. Diffuse dissemination of a cerebellar medulloblastoma in the spinal arachnoid space.

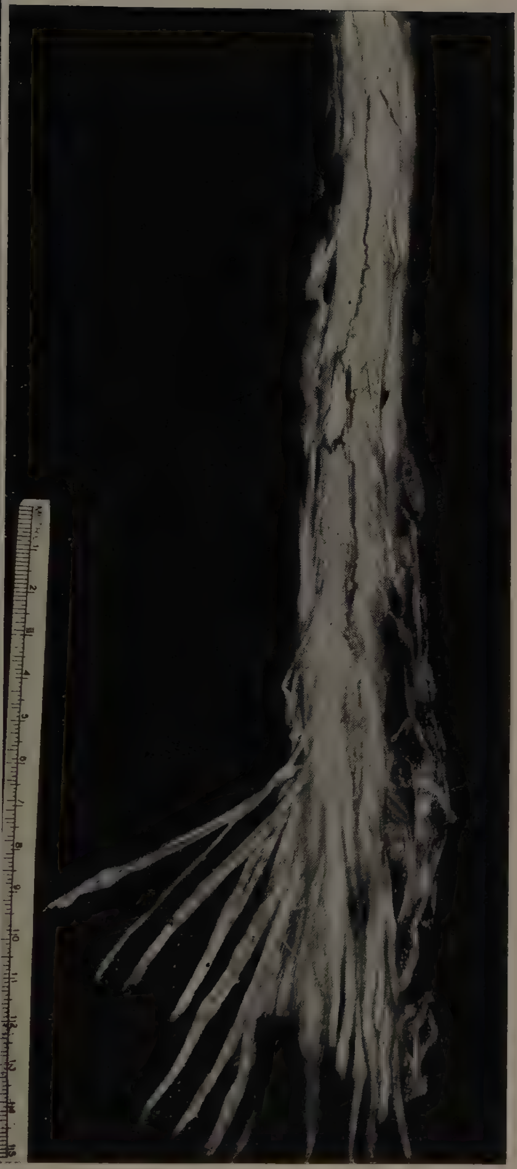


FIG. 166. Note the diffuse subarachnoid implantation of a cerebellar medulloblastoma about the lumbar and sacral spinal cord segments. There is diffuse and nodular thickening of the roots of the cauda equina.

glioma in past years in this laboratory. In 7 of these 11 cases, there are numerous diffuse and nodular implantations of tumor tissue in the spinal subarachnoid space from primary cerebellar medulloblastomas (Fig. 164). In two instances, astrocytomas of the brain stem had invaded the subarachnoid space and spread down about the cervical cord as single masses by direct extension, and an ependymoma of the fourth ventricle did the same. A pinealoma had given rise to a single implant in the cervical subarachnoid space.

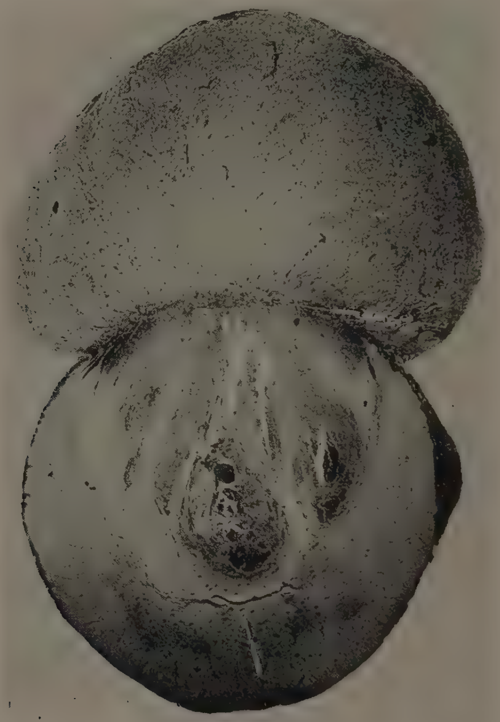


FIG. 167. A secondary mass of medulloblastoma from a cerebellar tumor is implanted in the thoracic leptomeninges and invades the spinal cord. Note the extensive extramedullary mass on the dorsal aspect of the cord (T 1), sharply marginated and extensively fibrosed. There is an intramedullary mass invading and destroying the posterior columns, and compressing both the gray and the white matter. Hematoxylin eosin stain. ×15.

The predominance of medulloblastomas among such secondary spinal gliomas is striking and is such as is described in other glioma series. In all 7 instances, tumor implants are scattered throughout the spinal subarachnoid space including the cauda equina region

(Figs. 164, 166), where they are often particularly frequent. They are predominantly dorsal in position (Fig. 164), although they may completely surround and encase the cord. They are usually very numerous, irregularly distributed, often cluster and coalesce, and frequently invade the underlying spinal cord (Fig. 167). They may be diffuse and produce cream-colored or grayish discoloration, opacity and thickening of the leptomeninges (Fig. 165). More commonly they are nodular, white, grayish white or cream-colored, usually firm, although they may be soft, distend the subarachnoid space, compress and distort the spinal cord and produce an irregular swollen appearance of the spinal cord and leptomeninges (Fig. 164). These nodules may vary from some the size of a pinhead to others the diameter of the cord itself or larger. On section, the portion in the leptomeninges is often found to be firm, tough, and white, while that invading the parenchyma is more cream-colored, granular and soft. The spinal cord proper, where compressed or invaded,

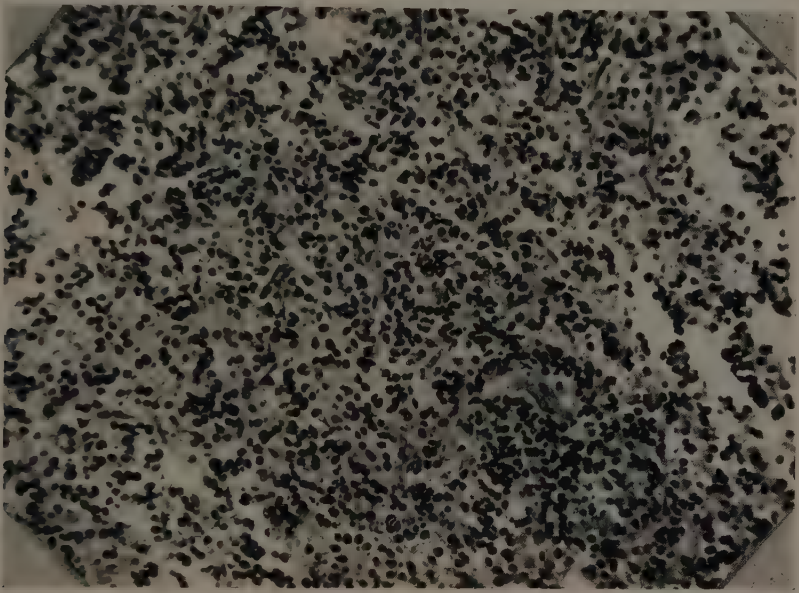


FIG. 168. A secondary implant from a cerebellar medulloblastoma invaded the posterior columns of the spinal cord at T 1. The tumor cells are small and have hyperchromatic, spherical uniform nuclei. The cells are arranged in pseudorosette formation. Hematoxylin eosin stain. ×230.

shows varying degrees of diffuse myelomalacia. In one instance there is a lumbar syringomyelia where a dorsal subarachnoid tumor mass, which invades the cord, is present. In another instance, there is a syringobulbia in the presence of subarachnoid tumor which surrounds the brain stem and spreads down about the cord.

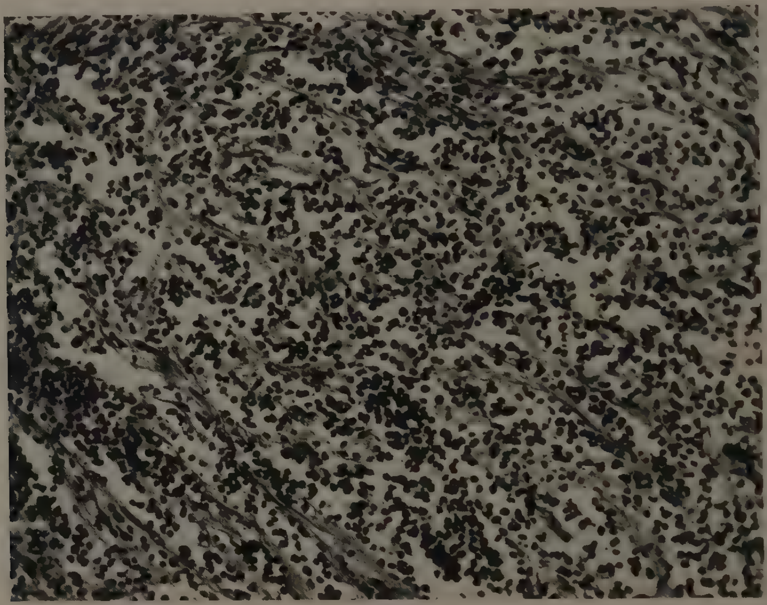


FIG. 169. A medulloblastoma invaded the leptomeninges on the dorsal surface of the spinal cord at T 1. Note the fibrous tissue of leptomeningeal origin, forming trabeculae between clusters of tumor cells. Hematoxylin eosin stain. ×230.

While the portion of these spinal metastases which invades the spinal cord is histologically identical with the primary cerebellar neoplasm (Fig. 168), the part of the implant in the spinal leptomeninges often shows some differences. The small rounded, oat or carrot-shaped, scant-bodied tumor cells in the latter situation are only rarely arranged in pseudorosettes or characteristic clusters (Fig. 169). In addition, there is usually a marked reactive fibrosis in the invaded leptomeninges so that the cells are embedded in a rich connective tissue stroma, an appearance which may simulate that of a sarcoma (Fig. 169).

Three intracranial gliomas reach the spinal subarachnoid space by direct extension. Two are astrocytomas of the brain stem, one being composed chiefly of protoplasmic and the other of fibrillary astrocytes of the piloid variety. Each invades the subarachnoid space about the medulla and extends caudally about the upper cervical spinal cord segments. The first is grayish and soft, and the second grayish white and solid, and each appears encapsulated because it is covered by leptomeninges. In neither instance is the spinal cord itself invaded. In one case there is an associated syringobulbia and a cervical syringomyelia. The ependymoma of the fourth ventricle has extended caudally into the cisterna magna and along the subarachnoid space over the dorsal surface of the spinal cord to the level of the third cervical segment. The subarachnoid mass is reddish gray and quite soft. It reproduces the features of the primary fourth ventricle tumor. Groff (1937) collected instances, chiefly intracranial, of such dissemination of gliomas by direct extension. The pinealoma is a single implantation in the subarachnoid space on the ventral surface of the third to fifth cervical spinal cord segments

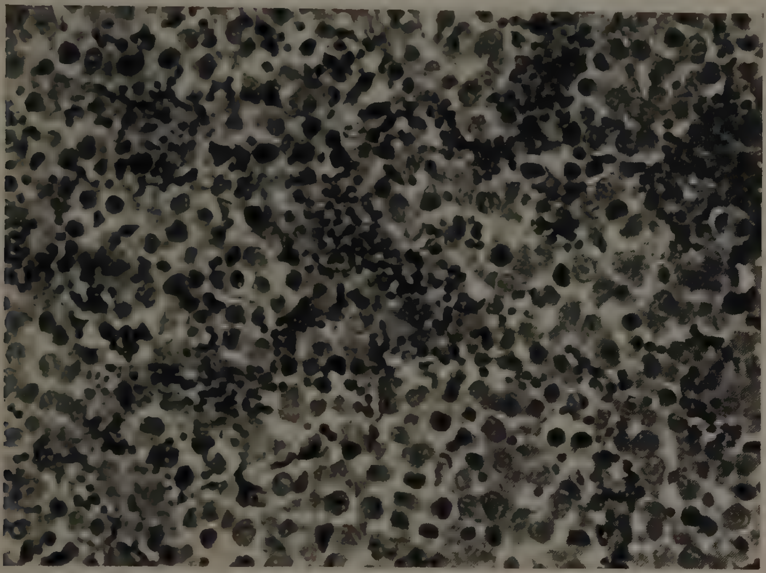


FIG. 170. A secondary pinealoma in spinal leptomeninges at C 3 to C 5. Two types of cells are represented. There are large, rounded or polygonal cells with very large, prominent, spherical or oval nuclei filling most of the cell body, having coarsely granular chromatin and small cells with scanty cytoplasm and small hyperchromatic spherical nuclei. (Many of the large cells have eosinophilic intranuclear inclusions in this instance.) Hematoxylin eosin stain. ×300.

from a primary pinealoma which had been successfully removed four years previously. It invades the dura secondarily. The implant is quite cellular and fairly vascular. The cells are roughly subdivided into small islands by a vascular network. The chief neoplastic element is a large, polygonal cell with a moderate amount of finely granular, eosinophilic cytoplasm. The nucleus is very large and prominent, filling a good part of the cell, and containing a great deal of finely granular chromatin (Fig. 170). Many of the nuclei contain varying-sized eosinophilic inclusions surrounded by halos. Between the cells there is a fine meshwork of cytoplasmic processes which can be traced to some of these cells. In addition to these large cells, there are focal collections of small lymphocyte-like elements with scanty cytoplasm and spherical, hyperchromatic nuclei (Fig. 170). Hyperplastic connective tissue cells and a network of reticulin and collagen of leptomeningeal origin are present. This leptomeningeal fibrosis, already mentioned a number of times, is seen in varying degree in all instances of neoplastic invasion of the leptomeninges. Usually clusters or large islands of tumor cells are surrounded by connective tissue trabeculae, but occasionally single rows of cells or individual tumor elements are separated by connective tissue fibers.

SECONDARY IMPLANTATIONS OF OTHER INTRACRANIAL TUMORS IN THE SPINAL SUBARACHNOID SPACE

Cairns and Russell (1931) described three small spinal subarachnoid metastases of a large, very soft pituitary adenoma (type not stated). One tiny metastasis was present at the extreme tip of the cauda equina and the other two were on the dorsal surface of the spinal cord.

Bassoe (1916), Schuberth (1926), Cushing (1932), and Bailey (1933) described instances of *diffuse primary sarcomatosis* of the leptomeninges of the brain, chiefly basilar, with extension down into the spinal subarachnoid space. In these cases, the meninges appeared thickened and opaque. The tumors were highly cellular and contained large numbers of reticulin fibers, closely related to the tumor cells. The neoplastic elements were lymphocyte-like or elongated, but most often they were polygonal or rounded cells, having abundant acidophilic cytoplasm and eccentric oval nuclei. Mitoses were frequent. *Primary malignant melanoma* or melanoblastoma occurring in the leptomeninges at the base of the brain might either extend into or occur simultaneously or primarily in the spinal leptomeninges, (Foot and Zeek, 1931; Bailey, 1933; Akelaitis, 1935; Wilcox, 1939). Many of the primary leptomeningeal melanosarcomas reported, however, were doubtful and probably secondary.

Cairns and Russell (1931) presented an interesting table of cases collected from the literature as well as those personally observed which were examples of the association of intracranial tumors and intraspinal lesions.

Brain	*Spinal Cord*
1. Primary cerebellar glioma	Secondary glioma, by extension in continuity through foramen magnum.
2. Primary cerebral glioma	Metastatic spinal glioma
3. Metastatic cerebral glioma (from glioma of retina)	Metastatic spinal glioma
4. Metastatic cerebral glioma	Primary spinal glioma
5. Sarcomatosis of meninges	Sarcomatosis of meninges
6. Primary cerebellar angioma (hemangioblastoma)	Primary spinal angioma (hemangioblastoma)
7. Primary intracranial meningioma	Primary or ? metastatic spinal meningioma
8. Neurofibromatosis of roots of cranial nerves	Neurofibromatosis of roots of spinal nerves
9. Combinations of 7 and 8	With or without spinal glioma
10. Primary melanomatosis	Primary melanomatosis
11. Pituitary adenoma	Metastatic pituitary adenoma
12. Metastatic carcinoma or sarcoma	Metastatic carcinoma or sarcoma
13. Primary intracranial tumor	Syringomyelia

SYRINGOMYELIA AND SPINAL CORD TUMORS

Autopsy material was available in 11 of the 37 instances of primary and secondary intraspinal glioma in the present series. In 3 of the 11 cases, a syringomyelia was present, which seemed to be related to the neoplasm. Among 4 cases of intraspinal hemangioblastoma to be discussed later, only one was examined post mortem, and this showed a syringomyelia associated with multiple hemangioblastomas. Thus, in a group of 12 spinal neoplasms examined post mortem, 4 showed this association. Kernohan (1932) had autopsy material in 10 of 67 instances of primary tumors of the spinal cord or filum terminale. Excluding a tuberculoma, 5 of the remaining 9 neoplasms were associated with syringomyelia.

Of our 4 cases of spinal cord tumor with syringomyelia, one is an upper cervical astrocytoma, another is an ependymoma of the filum terminale which spread upward in the spinal subarachnoid space, a third, a cerebellar medulloblastoma metastasis in the spinal subarachnoid space which invaded the spinal cord, and the fourth is the instance of multiple spinal hemangioblastomas in a case of Lindau's disease to which reference has already been made.

The upper cervical astrocytoma was described in the section on astrocytomas. It invades the subarachnoid space and spreads upward about the brain stem, cerebellum, and base of the brain. A syrinx extends in the cephalic direction from the tumor, occupying the ventral portion of the posterior columns, left posterior horn and part of the left lateral column of the upper cervical cord, and enters the lower medulla where it is situated dorsal to the obliterated central canal. The caudal end of this continuous syringomyelia and syringobulbia is surrounded by tumor but the rest of the wall is composed of a dense band of glial tissue.

As described in the section on ependymomas, the ependymoma in the cauda equina region does not invade the spinal cord at any point. The leptomeningeal arteries, however, where the tumor massively invades the subarachnoid space on the right side of the lumbar cord, show a remarkable obliterative endarteritis (Fig. 153) and medial calcification. An area of edema, degeneration, and astrocytosis leads from the right lateral and posterior columns beneath the subarachnoid tumor in the region of the fifth lumbar segment to a cavitation in the posterior columns and commissures dorsal to the patent central canal. This area has the typical appearance of a syringomyelia (Fig. 151) and spreads upward to the second lumbar segment. The wall consists of a narrow band of gliosis and it is not lined by ependyma (Fig. 152). The central canal is displaced ventrally. The adjacent anterior and posterior horns are edematous (Fig. 151), cribriform, and show marked astrocytosis. A column of edema, degeneration, and astrocytosis spreads upward through the ventral portion of the dorsal columns, the dorsal commissures and portions of the gray horns as high as the third thoracic segment. Disseminated ependymoma implants are encountered in the subarachnoid space as high as the third thoracic segment.

The metastatic medulloblastoma is one of many dorsal implants in the spinal subarachnoid space, but it is by far the largest, measuring 2.5 by 1.5 by 0.5 cm., and is located in the midlumbar region. Tumor invades the cord and there is a secondary syringomyelia which is purely lumbar but extends in each direction above and below the nodule. Tumor does not reach the wall of the cavity. The latter is quite similar in type and position to the cavities described above.

In our case of Lindau's disease, there are three intraspinal hemangioblastomas. One of these is bedded deeply in the posterior columns, high in the cervical cord; another occurs in a posterior horn of the lower thoracic cord, and the last is attached to the cauda equina. Above the high cervical tumor, a syringomyelia in the typical position in the posterior columns (Fig. 176), dorsal to the obliterated central canal, with finger-like canals burrowing into the gray matter, is present in the first cervical segment, and extends in the cephalad direction to become a syringobulbia of the lower half of the dorsal aspect of the medulla. Caudal to the tumor, a syringomyelia, sometimes multiloculated, extends from the fifth cervical to the second thoracic segment. Edematous, degenerated parenchyma which has undergone gliosis extends from the margins of the neoplasm, both above and below the tumor, to the respective cavities described. Caudal to the tumor in the tenth thoracic segment, a cavity extends downward to the fourth lumbar segment in the posterior columns and commissures just dorsal to the central canal. The walls of all of the cavities are composed of a dense mass of glial fibers which is loose-meshed internally where small numbers of shrunken, old, fibrillary astrocytes are present; and more compact externally where moderate numbers of hypertrophied, full-bodied fibrillary astrocytes are found (Fig. 177).

Kernohan's (1932) 5 instances of spinal cord tumor with syringomyelia included 1 primary medulloblastoma, 1 oligodendroglioma, 2 ependymomas and 1 hemangioblastoma. The associated syringomyelia was described as not unusual in type. The cavities were often extensive, surrounded by gliosis, non-neoplastic in type, and not lined by ependyma. They lay dorsal to the obliterated central canal. Kerkwitz (1934) reported a small circumscribed intramedullary glioblastoma with longitudinal cavitation extending several segments above and below the tumor. There was only a mild gliosis at its margin, the cavity being evidently of recent origin. Lichtenstein and Zeitlin (1937) described a syringomyelia associated with an intramedullary ganglioneuroma and referred to it as a "pseudosyringomyelia," distinguishing it from idiopathic syringomyelia by various histological criteria which seem to us to be of minor significance. That syringomyelia might be associated with intramedullary tumors other than gliomas was evidenced in the series of 12 spinal hemangioblastomas, referred to by Wolf and Wilens (1934), of which 7 showed an associated syringomyelia. Antoni (1936) called attention to the fact that extramedullary and even extradural tumors may be associated with syringomyelia, and cited an extramedullary lipoma (Bielschowsky and Valentin, 1927), an intradural meningioma (Antoni, 1920, Case 24), a sarcoma of the dura (Kronthal, 1889), and an endothelioma of the vertebra (Alexandroff and Minor, 1896). Both intramedullary and extramedullary intraspinal teratomas may be associated with syringomyelia (Gerlach, 1894; Bielschowsky and Unger, 1920; and Voss, 1938).

The association of syringomyelia with spinal cord tumors has been explained in various ways. Lindau was of the opinion that these cavities, in the cases of spinal hemangioblastomas were simply elongated cysts similar to the cysts so commonly encountered about cerebellar hemangioblastomas and due to transudation from the vessels of the tumor. He believed that they showed only a superficial resemblance to true syringomyelia, however. The circulatory disturbances in vascular tumors, he thought, caused edema, which spread in the neural parenchyma about the tumor. The spread of edema fluid occurred as did that of the blood in hematomyelia, in narrow irregular columns up and down the cord, usually for long distances. Such edema of the tissues is quite marked in all of our four cases cited in the foregoing. Where the fluid collected, cavities formed, the nervous and glial tissues being compressed and undergoing degeneration. The pressure of the fluid and the necrosis gave rise to a reactive gliosis composing the walls of the syringomyelic cavity. As we have seen, however, syringomyelia occurs with other than vascular tumors, just as do cerebellar and cerebral cysts. Among our own and Kernohan's cases are ependymoma, astrocytoma, oligodendroglioma, and primary and secondary medulloblastoma of the spinal canal. Lichtenstein and Zeitlin (1937) reported a ganglioneuroma and Berkowitz (1934) a glioblastoma. It may be that pressure of the expanding lesion results in a circulatory disturbance in these instances as well. This is strikingly indicated in our case of ependymoma of the cauda equina region; the tumor invades the subarachnoid space but not the spinal cord. Where the largest subarachnoid metastasis is present, to the right of the lumbar cord (but actually mostly on its dorsal surface since the cord is markedly rotated), there is a localized obliterative endarteritis obviously secondary to the tumor compression. In this region there is a well-marked syringomyelia. In every instance, the cavities in the cord course away from the level of the tumor, extending both above and below it.

Kirch (1928) pointed out that in addition to long columns of edematous, partially degenerated tissue undergoing gliosis, solid gray glial columns, such as were considered to antedate the cavity in true syringomyelias, could be found in relation to intramedullary cord tumors. This is also true in the cervical region in our case of extramedullary ependymoma. Kirch suggested that in these cases no collections of edema fluid have formed, a more diffuse infiltration of the tissue by serous transudate having occurred. This seems unlikely from our example, in which the edematous column is continuous with the solid glial one. Tannenberg (1924) believed that softenings occurred in the region of a cord neoplasm with resultant cavity formation and gliosis rather than mere transudation, fluid collection, and tissue compression. He considered the necroses to be due both to local circulatory disturbances and to toxins elaborated by the tumor. Russell (1932) likewise believed that anemic or hemorrhagic softenings due to the presence of a tumor in the cord may give rise to syringomyelia. She quoted Cornil and Ranvier for evidence that softening occurring after trauma followed the same distribution as hemorrhage in the cord. A column of softened tissue in the posterior columns and horns, parallel with the long axis of the cord, might extend for long distances. Finally a cavity with a glial wall was formed. That such necrosis, secondary to tumor might extend for some distance from it was shown by Russell's case of metastasis of a pulmonary carcinoma to the first and second lumbar segments with softening of the spinal cord up to the sixth thoracic segment.

Another explanation of the association of spinal cord tumors and syringomyelia is that they are coincident, multiple, congenital anomalies, at least in some cases. Bielschowsky and Unger (1920) expressed this opinion and Jonesco-Sisesti (1929), Mackay and Favill (1935), and Bailey and Foerster (1936) more recently, favored it. Bielschowsky and Unger, and Jonesco-Sisesti adduced the evidence of the conjunction of teratomas and teratoid tumors of the spinal cord with syringomyelia. Furthermore, the occurrence of syringomyelia with multiple congenital malformations, such as is seen in Lindau's disease, as was true in our case of multiple hemangioblastomas cited earlier in this discussion, or in neurofibromatosis, would be in favor of this theory.

A final possibility is that hemorrhage from intramedullary tumors, in particular hemangioblastomas, may produce hematomyelia which results in syringomyelia. Russell (1932) discussed this theory and pointed out that Ohlmacher's case of a cavernous angioma of the cervical cord was accompanied by a hematomyelia which extended for some distance above and below the tumor. She quoted a case of her own, an ependymal glioma of the lumbar cord associated with syringomyelia. There was evidence of an old hemorrhage into the borders of the syringomyelic cavity and into the walls of a cyst within the tumor. Russell believed that this might be the cause of the association in some cases, but that most often when there is hemorrhage, it is secondary to the circulatory disturbance and its resultant necrosis.

In our cases the syringomyelia is quite typical, although in three instances, it is not extensive, being in an early stage of development. Fundamentally, there seems to be no difference in the walls of these cavities from those of syringomyelia without tumor. What differences there are seem to be dependent on the stage of development of the cavity. In each instance the cavitation is intimately associated with the neoplasm. Markedly edematous areas, often with intense accompanying degeneration, are directly continuous with the syringomyelia and are contiguous to the tumors. They point to a local vascular disturbance.

From our own material, therefore, we have reason to believe that these cavities in the cord are comparable to so-called true syringomyelia, and that the most plausible explanation of their formation is that columnar edema and necrosis occur secondary to circulatory disturbances produced by the tumor, with subsequent cavity formation and mural gliosis.

VASCULAR TUMORS OF THE SPINAL CANAL

Cushing and Bailey (1928), in their monograph on blood vessel tumors of the brain, clearly differentiate between angiomas and hemangioblastomas of the central nervous system. Angiomas they classify as malformations composed of arteries and veins, stressing the point that nervous tissue in various states of preservation is always found between the vessels of these lesions when they are intraparenchymal. Hemangioblastomas they consider to be true vascular neoplasms. In addition, a mass of dilated capillaries with nervous parenchyma between the vessels or single dilated tortuous arteries or veins are separately grouped as telangiectases.

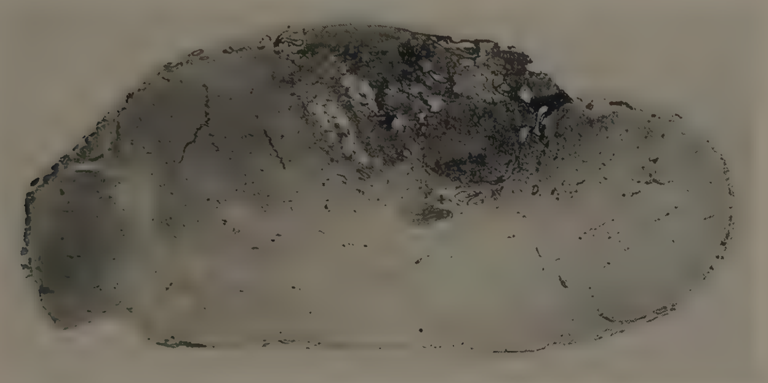


FIG. 171. A hemangioblastoma of the leptomeninges of the cervical enlargement of the spinal cord. The tumor is invading and compressing the posterior columns and one posterior horn. There is considerable edema, congestion and degeneration of the parenchyma. Hematoxylin eosin stain.

HEMANGIOBLASTOMA

There are 4 instances of intraspinal *hemangioblastoma* among the 253 intraspinal neoplasms in this series. Two of these are in cases of Lindau's disease, in 1 of which, mentioned previously, there are multiple tumors, 2 intramedullary and 1 attached to a root of the cauda equina. In 2 of the 4 cases, the neoplasm is both intraspinal and in the posterior fossa, one extending from the first cervical segment into the medulla, and the other being in the vermis of the cerebellum and extending into the spinal subarachnoid space over the first cervical segment. In one instance the neoplasm is extradural at the level of the second and third thoracic vertebrae, firmly attached to the left dorsal root.

Globus and Doshay (1929), in reviewing the literature, found 7 intramedullary hemangioblastomas of the spinal cord and 1 in the cauda equina. Kernohan (1932) in his group of 67 primary tumors of the spinal cord reported 4 instances of hemangioblastoma of the spinal cord and 3 of the filum terminale. One of the spinal cord cases was an instance of Lindau's disease, having two intramedullary neoplasms.

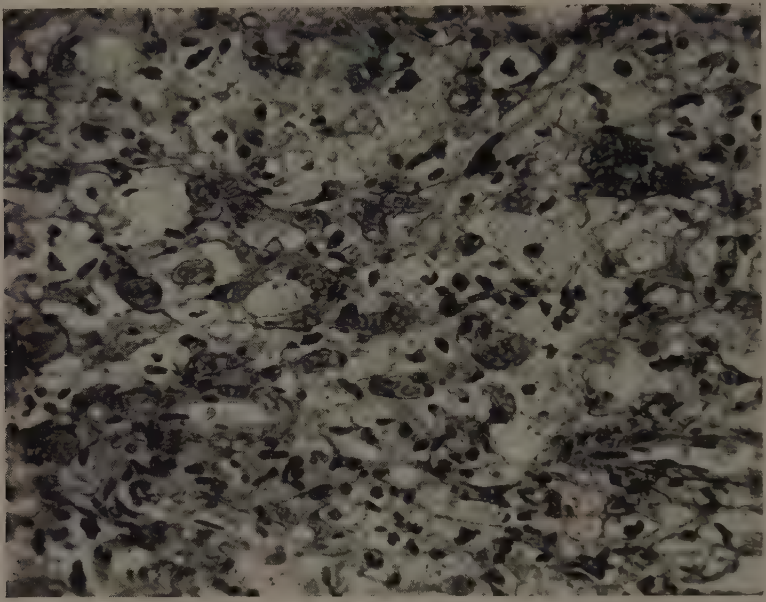


FIG. 172. Hemangioblastoma of the cervical cord. Many blood vessels of the order of capillaries are lined by plump endothelial cells having prominent nuclei. The intervascular cells are chiefly large foam cells with vacuolated, translucent bodies and small hyperchromatic nuclei-pseudoxanthoma cells. Hematoxylin eosin stain. ×230.

All of the spinal hemangioblastomas in the present series are dorsal in position (Fig. 171). They are yellowish brown and soft, or reddish or reddish purple and moderately firm. The intramedullary examples lie beneath the pia, merging with it, and are embedded deeply in the posterior columns and occasionally in the posterior horns (Fig. 171); the one that extends into the lower medulla is in a corresponding position in that structure. Viewed from the surface they may not be very conspicuous due to the fact that they lie almost flush with the external surface of the parenchyma. On section they are found to be fairly well demarcated. Histologically, all the hemangioblastomas in this series are quite uniform in appearance except for the single extradural example which is of the more cellular type. The predominant elements are blood vessels, which are capillary in type and fairly irregular in size and shape (Figs. 172, 174). Occasionally these lie closely approximated but more frequently they are separated by one or more layers of intervening cells (Fig. 172). Some larger vascular channels are also present but their walls are composed of collagenous connective tissue with no evidence of arterial or venous structure (Fig. 174). From these, the surrounding capillaries are often found to radiate (Fig. 175). The capillaries, as noted above, are quite irregular with much branching and outpocketing. They are lined by endothelial cells which vary from flat, elongated cells with oval, dark concentrated nuclei, to plump cells with oval, vesicular nuclei (Figs. 172, 173). These frequently seem to merge intimately with the intervascular elements. The capillary lumina are either empty or contain varying numbers of erythrocytes but no immature blood elements. The walls of the larger chan-

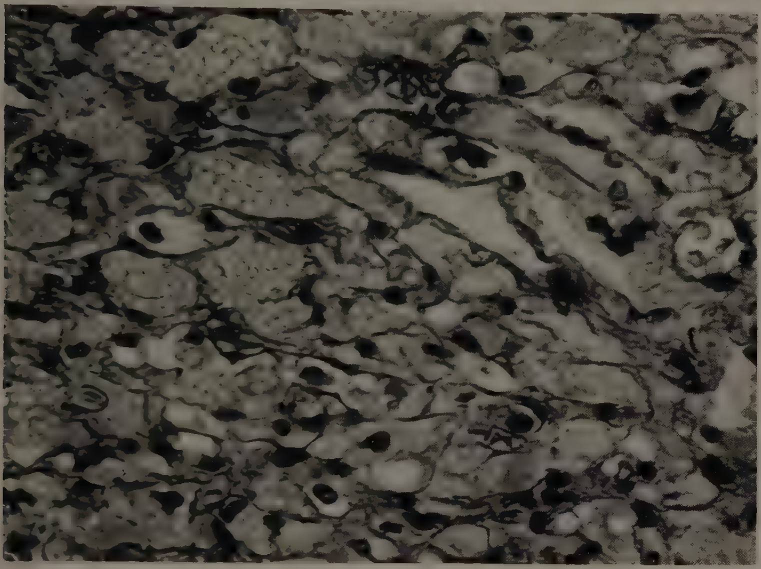


FIG. 173. Hemangioblastoma of the cervical cord. Note the pseudoxanthoma cells. These are large oval, rounded or polygonal cells with coarsely vacuolated cytoplasm containing neutral fat and lipoids, and hyperchromatic oval, spherical or irregular nuclei (indented by the fat vacuoles) which are often eccentric. Note many collapsed capillaries with their plump endothelial cells. Hematoxylin eosin stain. ×460.

nels described above are at times quite thick and occasionally hyalinized. The cellular elements found between the vessels are chiefly large-bodied, polygonal and spherical elements with vacuolated, foamy cytoplasm, so-called pseudoxanthoma cells (Fig. 173). The nuclei are comparatively small, concentrated, often compressed at the periphery of the cell and so lunate-shaped. In other cells the nuclei are more centrally placed and angulated, presenting concave surfaces due to compression by the lipoid globules. Where these lipoid-laden cells are particularly abundant and most heavily vacuolated, the capillaries between them are compressed, though numerous, and at first glance seem to form only an insignificant portion of the picture (Fig. 173). Among these large-bodied cells, are less obvious, elongated cells with oval deep-staining nuclei. These cells appear to be endothelial in type, and are occasionally seen to be intimately associated with the cells lining the capillaries. A few scattered, rounded cells, somewhat smaller than the vacuolated cells, are found to be multinucleated. Occasional small cysts filled with pink granular material are seen, as well as evidence of more general-

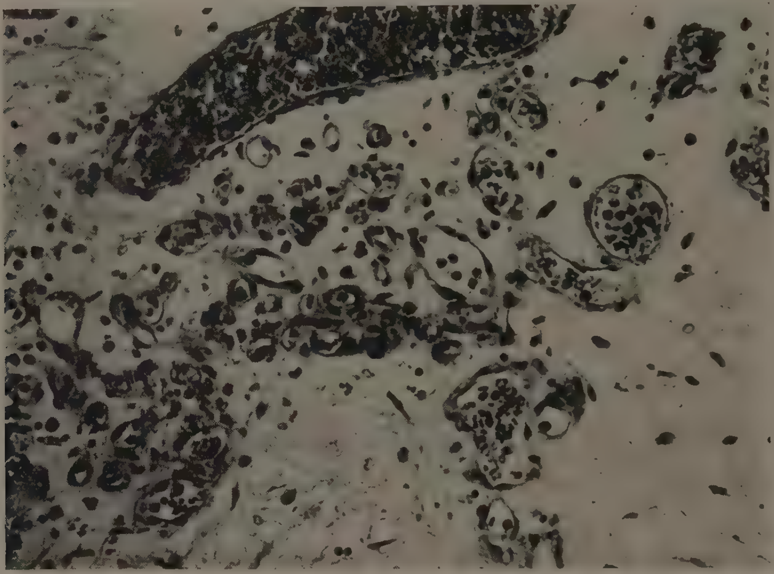


FIG. 174. Hemangioblastoma of the cervical cord. The varying sized capillaries are lined by plump endothelial cells. There is a thin-walled venous channel above. Hematoxylin eosin stain.

ized edema, such as the separation of individual cells by similar material. A very striking feature is an abundant network of reticulin fibers throughout the tumor outlining the capillaries, bridging between them and running to the collagenous walls of some of the larger vessels (Fig. 175). The intramedullary neoplasms, except for the deep lower thoracic tumor, are attached to the pia and are apparently growing in it at the surface. Internally, although the tumor has a definite margin, it has no capsule and numerous capillaries of the neoplasm extend beyond its general border into the surrounding neural tissue. The edema, degeneration and gliosis in the parenchyma near the tumors (Fig. 171) and the syringomyelia in one of the cases of Lindau's disease (Figs. 176, 177), have been described in the section on syringomyelia. The frequency of the association of cavitation of the cord with intramedullary hemangioblastoma has been emphasized.

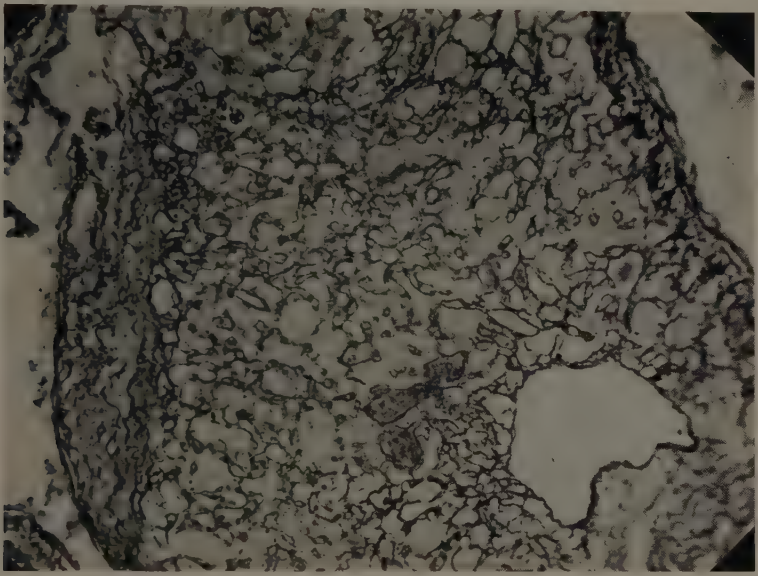


FIG. 175. Hemangioblastoma of the cervical cord. Note the reticulin network in the tumor, outlining the walls of the capillaries and showing their relationship to a larger venous channel on the right. Note also the intercapillary reticulin fibers frequently running from one vessel to another. Laidlaw connective tissue stain. ×110.

It is clear that hemangioblastomas of the central nervous system are uncommon. They comprise somewhat less than 1 per cent of intracranial tumors and perhaps 1 to 2 per cent of intraspinal neoplasms. They appear to be true neoplasms, capable of growth, which is sometimes fairly rapid. They do not spread along the ventricular system or subarachnoid space, the instance described being a rare exception. A number of things point to a congenital factor in the development of these neoplasms. One is their chief position in the condition known as Lindau's disease in which occur congenital lesions, such as pancreatic cysts, malformation and tumor of the kidney, adrenal, liver, and in which the disposition to angiomatosis of the eyes and central nervous system is often familial. Another is their frequent occurrence in the dorsal midline. Bielschowsky and Unger (1920), in discussing a teratoma of the cord, pointed out the possibility that portions of the surface membrane might be drawn down into the neural parenchyma during closure of the medullary groove. This might later give rise to mesodermal growths.

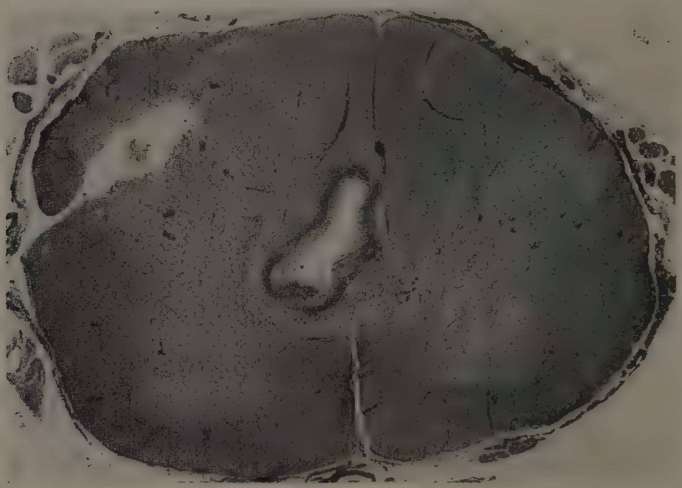


FIG. 176. Syringomyelia. This is in the lumbar cord, caudal to an intramedullary hemangioblastoma. Note the typical position of the cavity. Phosphotungstic acid-hematoxylin stain.

A group of supratentorial tumors, which resemble meningiomas grossly, and which histologically show many gradations from meningioma to hemangioblastoma, have been described as angioblastic meningioma by Bailey, Cushing and Eisenhardt (1928), and leptomeningioma and pial (vascular) meningioma, hemangio-endotheliomatous type by Globus (1935). No examples of this tumor intergrade have been described in the spinal canal, although some of our meningiomas have had highly vascular areas. It is suggested by Globus that these tumors arise from the pia from which he also derives the pure hemangioblastomas. The intimate association of our intramedullary hemangioblastomas with the pia would be in consonance with this suggestion. The cauda equina nodule might also have such an origin but the extradural tumor is difficult to explain on this basis.

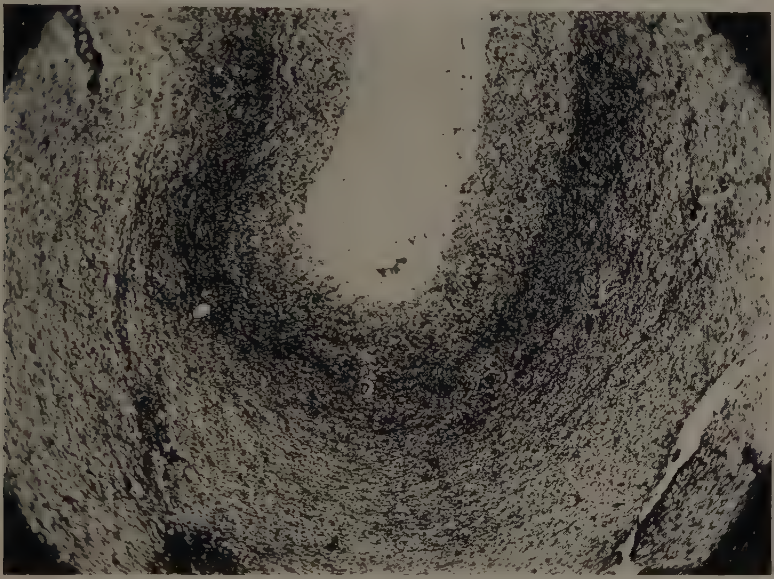


FIG. 177. A dense band of gliosis forms the wall of a syringomyelic cavity in the cervical spinal cord in the presence of multiple hemangioblastomas of the cord. The cavity has no ependymal lining. Phosphotungstic acid-hematoxylin stain.

ANGIOMA

Angiomas are at present considered by most pathologists to be developmental vascular anomalies, capable of subsequent alteration in size and character. They are relatively infrequent in the central nervous system and its envelopes. There are 14 examples among our 784 intracranial neoplasms. Five tumors of this type are included among the 253 intraspinal neoplasms of this series. One of these was leptomeningeal and intramedullary, 2 were extradural, 1 was extradural and vertebral, and 1 was purely vertebral with an associated osteoporosis and deformation of the vertebra, resulting in narrowing of the spinal canal. The intramedullary tumor was dorsolateral, subpial, at the level of the seventh thoracic segment, and occupied or compressed the major portion of the cord in this region, reducing it to a narrow ventral crescentic band. The tumor is rounded, red and soft but contains some firm gray areas. The 2 extradural angiomas were at vertebral levels T 4 to T 8 and T 5 to T 7, respectively. They are elongated, flattened and oval, dark red or reddish blue, and soft. The extradural portion of one vertebral angioma has the same appearance, while the vertebra proper is somewhat bluish and porous in appearance. This is also true of the other purely vertebral lesion occupying T 7 and T 8.

When seen at operation, the type of angioma present may be recognized by its vascular connections, and by the character of the blood in its vessels. The majority of these tumors are found to be venous, and the remainder, arteriovenous. The latter are usually derived from the venous angiomas through the development of arteriovenous fistulae, followed by arteriolization of some of their vessels, as suggested by Cushing and Bailey (1928). Although direct observation of these lesions during life permits their ready identification, histological classification is often difficult due to the malformation of the walls of the vessels composing them, so that arteries and veins can no longer be distinguished.

The leptomeningeal and parenchymal angioma previously referred to is composed of vessels which vary markedly in size and shape and present numerous pockets and pouches (Fig. 178). The majority have large lumina and walls of varying thickness. In most, the wall is narrow and composed principally of collagenous connective tissue lined on its inner surface by a layer of endothelial cells. In some, incomplete development of three layers may be seen. Media and internal elastic membrane may be present on one side of a vessel, and incompletely developed or absent on the other. The intima in a few vessels is markedly thickened, and some of the large vessels are thrombosed and the thrombi in varying stages of organization. In one vessel, a small myoma of the abnormal media is seen.

There is no mural calcification. The vessels for the most part lie closely approximated; when their walls are markedly thickened, fibrosed and hyalinized, and the vessels are occluded, sheets of dense fibrous tissue are formed. In the leptomeninges, patches of fairly well preserved pia-arachnoid are encountered, but for the most part, they have undergone thickening and fibrosis (Fig. 178). Hemosiderin-laden phagocytes are present in them, as they are elsewhere between the abnormal vessels. In deeper portions of the tumor, small, scattered islands of parenchyma are seen, in which the neural elements have completely disappeared and in which there is an intense gliosis (Fig. 179). Some of the vessels in this tumor would seem to be malformed arteries, and the tumor is, therefore, classed as an arteriovenous angioma.

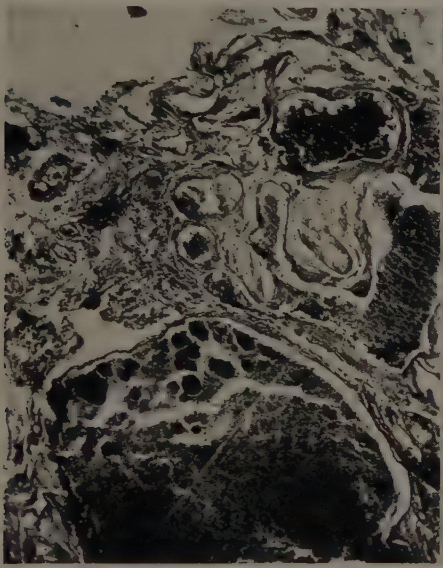


FIG. 178

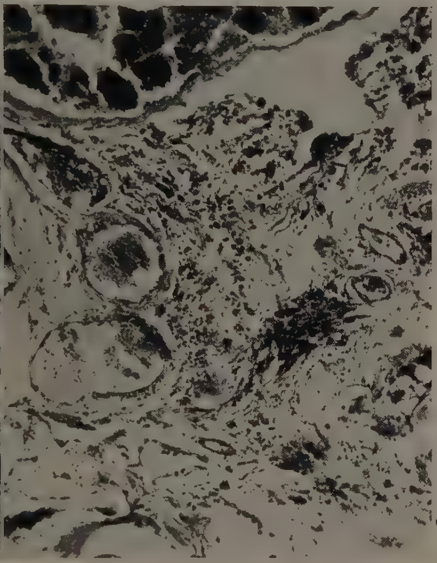


FIG. 179

FIG. 178. An angioma in the spinal leptomeninges and cord. Note the varying size of the blood vessels and the abnormal variation in the development of the vessel walls. The meninges are thickened. Hematoxylin eosin stain. ×60.

FIG. 179. Angioma in the spinal cord parenchyma. Hemosiderin-laden phagocytes are seen in the interstitial tissue. A darkly staining island of compressed fibrillary astrocytes represents a remnant of degenerated parenchyma which has undergone gliosis. Phosphotungstic acid-hematoxylin stain. ×60.

In the two extradural angiomas the vessels are less variable in type. They are frequently very thin-walled (Fig. 180). The vessels are often intimately interspersed with the epidural fat (Fig. 180). Of the vertebral angiomas, one shows the same variability seen in the intradural example, while the other is more uniform, like the extradural angiomas (Fig. 181). There is considerable bone atrophy of the trabeculae about the vessels in both instances with some attempt at bone regeneration. The vessels occupy the enlarged marrow spaces in which no marrow is seen (Fig. 181). No telangiectases or varicosities of the spinal leptomeninges are present among our pathological specimens. These are simply single abnormal vessels or small clusters of vessels, as mentioned earlier.

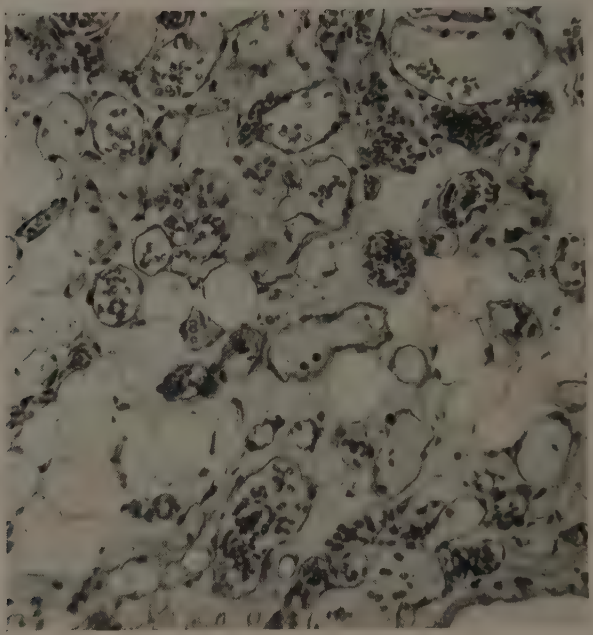


FIG. 180. Angioma. The neoplasm was extradural from T 4 to T 8. There are numerous thin-walled vessels of varying size in the epidural fat. Hematoxylin eosin stain. ×230.

The nature of angiomatous lesions has been variously interpreted. Virchow (1863) was of the opinion that they were neoplasms, and Ribbert (1898) later supported him in this view. Enders (1908) regarded them as neoplastic malformations, while Borst (1913) considered them to be simple anomalies. Others extended the latter view, describing the angiomas as congenital malformations possessing the potentiality of further growth and development. Cushing and Bailey (1928) and Dandy (1928) favored this interpretation, and it seems the most likely explanation. There are obvious intergrades between a single malformed, dilated, tortuous, misplaced vessel and the full-blown angioma of the leptomeninges spreading into the parenchyma in the form of a pyramid with its base in the leptomeninges. The often reported association with congenital nevi (port-wine nevi, hemangiomas) of the skin of the same or adjacent metameres, indicates the operation of a congenital factor in the inception of these lesions. Some of the spinal examples of this association are those of Cobb (1915), Kaplan (1935) and Johnston (1938). Cushing and Bailey (1928) were able to gather from the literature only 17 instances of spinal cord angioma, which indicates the comparative rarity of the lesion.

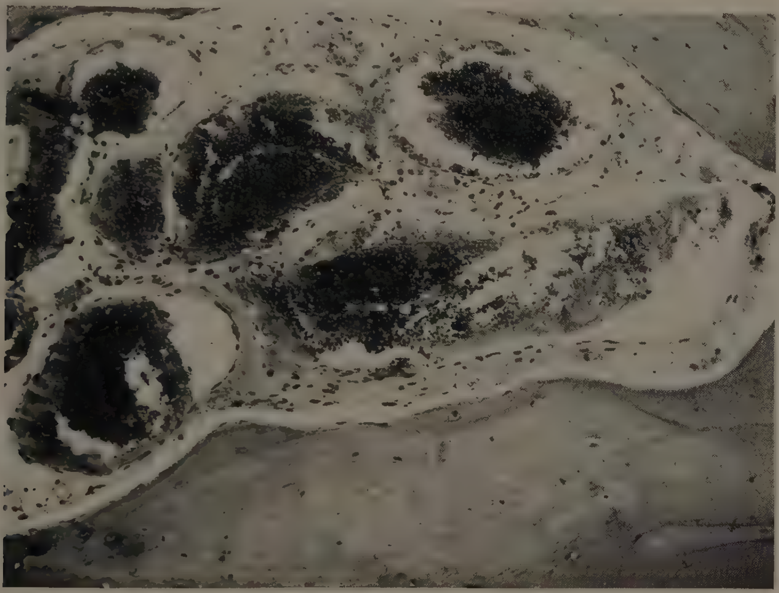


FIG. 181. Hemangioma of the vertebra at T 10. Large thin-walled vascular channels of varying size, whose walls show different degrees of maldevelopment, completely fill the marrow space and give rise to bone atrophy and new bone formation. Hematoxylin eosin stain. ×110.

Evidence of minor hemorrhage in and about an angioma is almost constant, as marked by the presence of hemosiderin. Large terminal leptomeningeal and parenchymal hemorrhages are common among the intracerebral angiomas (Wolf and Brock, 1935). These also occur in association with angiomas of the spinal cord as a hematomyelia (Buckley, 1936; Richardson, 1937) and as subarachnoid hemorrhage (Robertson, 1938, and Johnston, 1938).

CONGENITAL TUMORS

Chordoma

Small, gelatinous excrescences, projecting from the basisphenoid of the skull, were first described by von Luschka (1856), and Virchow later named them ecchordosis physaliphora. Müller (1858) suggested that they were remnants of the notochord, since they had the appearance of embryonal cartilage, and Ribbert (1895) proved this and proposed that tumors arising from them be called chordomas. Similar persisting notochordal tissue may be found in the nucleus pulposus of the intervertebral discs and as heterotopic vestiges at the cephalic and caudal extremities of the spinal column. In spite of this wide distribution of such remnants in relation to the spinal canal, spinal chordomas occur predominantly in the sacrococcygeal region and only rarely at other levels, the cervical tumors being next in frequency.

Two chordomas are present among the 253 intraspinal tumors of this series. One of these was at the level of the first and second sacral vertebrae, where it was extradural and extended through the dura to become intradural at the level of the fourth and fifth lumbar vertebrae. The intradural mass is small and bluish; the extradural is larger, reddish brown, soft and well encapsulated. The second chordoma was extradural at the level of the second to the fourth cervical vertebrae. It is flat, reddish gray, and encapsulated. After removal and recurrence three years later it was larger, penetrated the ventrolateral surface of the dura, and formed an intradural mass which indented the spinal cord from C 1 to C 5. The extradural portion is reddish blue and encapsulated; the intradural part dark red, mushy, and gelatinous. The tumor also invaded the body of the second cervical vertebra, which was eroded, resulting in widening of the spinal canal.

In the series of 557 intraspinal neoplasms of Rasmussen, Kernohan and Adson (1940), there were 23 chordomas. Of these 17 were sacral, 1 lumbar, 2 thoracic, and 3 cervical in position. Most of them had been found to invade and erode the vertebrae and fill the spinal canal, producing compression and degeneration of the spinal cord and nerve roots.

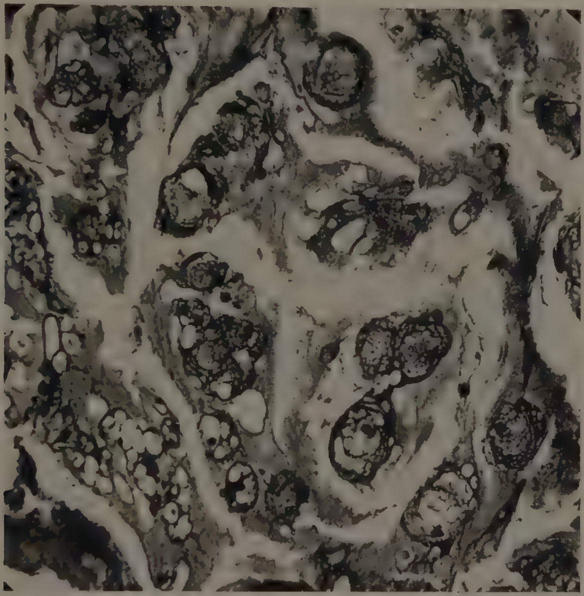


FIG. 182. Chordoma (sacrococcygeal). There are clusters of large bodied cells with coarsely vacuolated cytoplasm (physaliphorous elements). The nuclei are small, oval or spherical, hyperchromatic and at time vacuolated. The vacuoles frequently contain glycogen. Mucinoid intercellular material. Mallory's trichrome stain. ×300.

Histologically, the 2 tumors in the present series are quite characteristic and resemble tissue of the embryonal notochord. The neoplasms are rather weblike, or loose-meshed in structure. They are composed of cords, irregular columns or clusters of large, rounded, oval or polygonal cells referred to as physaliphorous cells, with coarsely vacuolated, pale, finely granular cytoplasm (Fig. 182). Their outlines are at times very vague. Some of the vacuoles are quite large and contain mucin or glycogen (Adson, Kernohan and Woltman, 1935, express doubt as to its being glycogen and believe it may be some related substance). The nuclei are oval, round, or polyhedral, comparatively small, hyperchromatic, and often eccentric. Their surfaces may be molded by the cytoplasmic vacuoles. These nuclei, too, are often vacuolated, and are reported to contain glycogen. Occasionally the cells are multinucleated. The intercellular substance is homogeneous and vacuolated or stringy (Fig. 182), has a bluish tint in the hematoxylin-eosin stain, and stains positively for mucin. The cell clusters may be separated by fine connective tissue trabeculae whose connective tissue cells are rather plump and fibroblastic in appearance.

These tumors may be locally invasive of bone and soft tissue; the bone was involved in one of our cases, and extensive local invasion was described in some of the instances of Fletcher, Woltman and Adson (1935) and others. Such tumors may, of course, originate in the bone itself. Owen, Hershey and Gurdjian (1932), in reporting 1 cervical chordoma and reviewing 8 others, pointed out that 1 arose from an intervertebral disc, 2 from the odontoid process of the axis, 4 from vertebral bodies, and in 2 the exact origin was unknown.

Epidermoid

Epidermoid (cholesteatoma, *tumeur perlée*). These pearly tumors are uncommon in both the intracranial cavity and the spinal canal. Our material contains 14 among 784 intracranial neoplasms, a figure somewhat higher than that for other series. Mahoney (1936), for instance, reported, that of 2500 verified intracranial tumors in the Cushing collection, only 15 were of this type. Only 1 of our 253 intraspinal neoplasms is an epidermoid, and Rasmussen, Kernohan and Adson (1940) reported none among 557 intraspinal neoplasms. Of 142 epidermoids collected by Mahoney (1936), only 7 were intraspinal.

The spinal epidermoid in our series was attached to the pia on the ventral surface of the spinal cord from the third lumbar to the first sacral segment.

The surface of this type of neoplasm is smooth, pearly white, silky and glistening, which accounts for the designation pearly tumor. It is tough and peels away easily or may show points of firm adherence. There is a thin, friable membrane over the pearly surface, reddish pink or gray in color (King, 1939). The neoplasm may have a nodular surface. It is malleable due to the softness of its cyst contents, which are white, caseous, and crumbly.

Histologically, the external surface of the tumor is found to be composed of a narrow band of connective tissue which, in this instance, is partially hyalinized and infiltrated with small numbers of lymphocytes and large mononuclear cells. Beneath this layer there is a band of stratified squamous epithelium of varying thickness, the stratum germinativum being outermost. Active keratinization is proceeding (Fig. 183). Keratohyaline granules are present in the cells of the stratum granulosum (Fig. 183). The epithelial elements become progressively more flattened and increasingly translucent as the stratum corneum is reached. The innermost tissue, and the most voluminous, is composed of the desquamated, keratinized epithelium, which appears as undulating parallel strips of semitranslucent material or is amorphous. Cholesterin crystals are present.

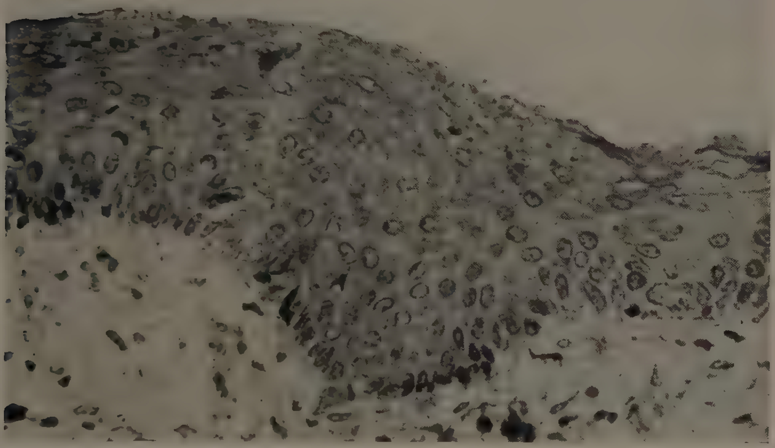


FIG. 183. Epidermoid cyst. The neoplasm was intradural from L 3 to S 1. The inner lining of the cyst wall is purely epidermal. Squamous epithelium lines the cavity and there is a loose layer of connective tissue external to it. Note the keratohyaline granules in the cells of the stratum granulosum, which are undergoing keratinization. Hematoxylin eosin stain. ×230.

The single intraspinal epidermoid in the series reported by King (1939) was attached to the roots of the cauda equina. The 7 intraspinal epidermoids among the 142 collected from the literature by Mahoney (1936) showed the following distribution: intramedullary D 4-6 (Chiari, 1883); cauda equina (Lauterburg, 1923); D 10-11 (Marinesco and Draganesco, 1924); D 7-8 (Critchley and Ferguson, 1920); D 5-6 (Melnikoff-Rasnedenkoff, 1931); L 2 (Schroder, 1932); D 11-12 (Michelsen, 1932).

These tumors apparently arise from misplaced rests of epithelial tissue in the leptomeninges, probably due to improper separation of neuroectoderm from surface ectoderm.

DERMOID

This is a neoplasm which contains not only epidermal elements like the preceding, but tissue of the corium or dermis, as well. It is rare in relation to the central nervous system. There are 13 examples among 784 intracranial neoplasms in our material, and 2 among 253 intraspinal tumors.

One of the intraspinal dermoids extended from the fifth to the seventh cervical segments, and was dorsal, intramedullary, extramedullary and extradural in position and extent. The other lay at the level of the fourth and fifth thoracic vertebrae and was dorsal, extramedullary, and extra-arachnoid, markedly indenting the spinal cord. The surface of these tumors, like that of the epidermoids, is lustrous, silky and pearl white; these tumors, too, are malleable because of their soft contents. Their central cysts contain white, cheesy material which differs from that in the epidermoids in containing hair.

Histologically, each tumor is found to contain material like that described in the large central cyst of the epidermoid. The inner lining of the cyst, here too, is stratified squamous epithelium, undergoing keratinization and desquamation, and so giving rise to the cyst contents (Fig. 184). External to the epithelial layer is a broad band of dense connective tissue in which hair follicles and sebaceous and sudoriferous glands are present (Fig. 184). In one instance, this layer shows considerable edema and some lymphocytic infiltration, and in the other, it contains a few smooth muscle fibers.

Gross (1934) gathered 19 epidermoid and dermoid tumors of the spinal canal from the literature, and added one; 10 (or possibly 11) were dermoids. Bradford (1937) collected 12 additional instances, of which 6 were intraspinal dermoids, and added an intramedullary example of his own. Of this total of 33 intraspinal epidermoids and dermoids, of which 16 were dermoids, there were 6 intramedullary epidermoids and only 3 intramedullary dermoids. Ten of the 33 tumors of both types occupied the region of the conus and cauda equina, and 2 dermoids were later reported in this region by Rasmussen, Kernohan and Adson (1940). The large dermoid reported by Trachtenberg (1898) in this region was associated with a great number of small epidermoid tumors disseminated throughout the cerebrospinal subarachnoid space and in the cerebral ventricles. A small dermoid of the right frontal lobe described by Raymond, Alquier and Courtellement (1904) was similarly accompanied by numerous, small, greasy masses scattered through the subarachnoid space, including the spinal. These small masses were said to stain poorly and their nature was not

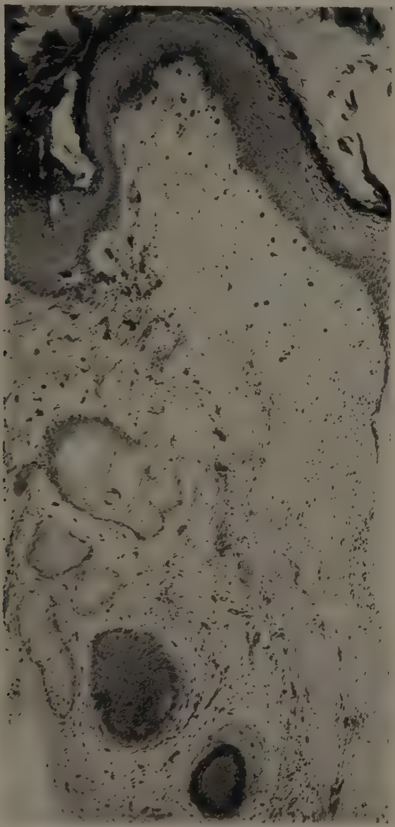


FIG. 184. Dermoid cyst. The neoplasm was intradural from T 4 to T 5. Squamous epithelium lines the inner surface of the cyst. Note the keratinization and desquamation of the lining epithelium producing the usual amorphous cyst contents. The dermal layer contains sebaceous glands and hair follicles. Eosin methylene blue stain. ×140.

further described, other than that they stained black with osmic acid and blue with hematin. The intramedullary dermoid of Ivanoff (1903) occurred in an anencephalic monster and extended throughout the spinal cord and medulla. Other intraspinal dermoids were associated with spina bifida or congenital dermal sinuses. In our case of dermoid of the thoracic spinal canal, T 4 and T 5, there was a telangiectatic area in the skin of the back in the midline of the T 6-7 dermatomes. The congenital nature of these tumors is further emphasized by the occurrence of such associated congenital lesions. Like the epidermoids, they are dysraphisms. The congenital dermal sinuses which may occur with them are of the same nature (Walker and Bucy, 1934). Either one or both may develop depending upon whether the surface ectoderm which has been invaginated with the neural ectoderm retains an epithelial connection with the surface.

Teratoma

Teratomas are composed of a disorderly mixture of tissues and organoid structures of bidermal or tridermal origin. The less complex examples are often designated as teratoids. These tumors are quite rare in the intracranial cavity and spinal canal, the teratoids being the more common (Hosoi, 1930). There are 2 teratoids among our 784 intracranial neoplasms and none among the 253 intraspinal tumors.

Rasmussen, Kernohan and Adson (1940) reported 1 extramedullary teratoma among 557 intraspinal neoplasms. Masten (1940) found that only 14 teratomas of the spinal cord had been reported, of which 2 were considered to be tridermal in origin. Bucy and Buchanan (1935), using the term teratoid to designate teratomatous tumors of bidermal origin (ectoderm and mesoderm), following Hosoi (1930), analyzed 10 such neoplasms. Masten (1940) believed that the ciliated epithelium in many of these tumors is respiratory, therefore entodermal, and that more than the 2 which were so reported were, therefore, tridermal in origin. Of the 11 intraspinal teratoids collected by Bucy and Buchanan, 3 were upper cervical, 1 midcervical, 1 cervical and thoracic, 2 lower dorsal and lumbar, 2 lumbar, 1 sacral in position, and 1 lay in relation to the conus medullaris. These tumors may be intramedullary or partly intramedullary (Gerlach, 1894; Bielschowsky and Unger, 1920), subarachnoid (Bucy and Buchanan, 1935), or subdural (Masten, 1940). It may be that some are continuous with extravertebral masses (Bucy and Haymond, 1932).

These tumors are chiefly cystic, although in some instances solid tissue may predominate. The cyst wall is thin and grayish, often translucent, and contains gelatinous, translucent or opalescent, grayish, thick, tenacious material. The solid areas are grayish white, elastic, and firm. Histologically, the cyst wall is lined externally by a narrow, loose-meshed, vascular connective tissue layer which is lined internally by a single layer of ciliated columnar or cuboidal, pseudostratified or even squamous epithelium. In the solid areas all mixtures of ectodermal and mesodermal and, more rarely, entodermal elements are encountered. The types of epithelium described above in the cyst wall, nerve cells, nerves, muscle, cartilage, bone, varieties of glands, and so forth are embedded in connective tissue. As pointed out by Ewing, there may be an overgrowth of one dermal element with malignant transformation of the tumor. This occurred in one of our two intracranial teratoids, one arising in the pineal, in which a carcinoma had developed.

Both intramedullary and extramedullary intraspinal teratomas may be associated with syringomyelia. In the cases of Gerlach (1894) and Bielschowsky and Unger (1920) the neoplasms bordered the cavity. Voss (1938) described an intradural teratoma over the caudal extremity of the spinal cord, with a syringomyelia and columnar gliosis extending from that zone to the seventh thoracic segment.

The commoner sacrococcygeal teratomas lying anterior or posterior to the sacrum, and the rare lumbosacral tumors similarly located (Bucy and Haymond, 1932) are not considered here.

Bailey and Buchanan (1935) correctly outlined the probable origin of teratomas. Unlike the epidermoid and dermoid which arise from an inclusion of surface ectoderm (and mesoderm), a simple mechanical embryological malformation, the teratomas and teratoids probably originate from a misplacement of multipotential germinal cells early in embryonic development and may depend upon a defect in the ovum.

Lipoma

The lipoma is a tumor composed of adult fat cells, and has the appearance of ordinary fat. It is not uncommon in relation to the central nervous system, being more common in the intracranial cavity. It may be single, multiple, or diffuse. It occurs not infrequently as a spinal extradural neoplasm but only rarely intradurally.

There are 4 lipomas among our 254 intraspinal neoplasms and 4 were also found among the 557 intraspinal tumors reported by Rasmussen, Kernohan and Adson (1940). Three intraspinal lipomas in our series are of the rare intradural type; 1 sacral example is associated with a spina bifida and meningomyelocele, in which the dural sac merges with the lipoma which enters the sac and surrounds the roots of the cauda equina. Of the intradural lipomas, 1 was high thoracic in position, 1 extended throughout most of the thoracic region, 1 surrounded the roots of the cauda equina, and all were dorsal. Each was intraleptomeningeal and the extensive thoracic neoplasm was largely intramedullary as well. They were well demarcated, lobulated, soft or firm, and canary yellow or grayish yellow in color. Histologically, they are composed of the large, polygonal, closely approximated translucent elements seen in normal adult fat.

The commoner extradural spinal lipomas are frequently associated with spina bifida, as is true in one case above; and some are said to be mixed tumors (Berenbruch, 1890; Liebscher, 1901), so-called angiolipomas. Cushing and Bailey (1928) have suggested that some of the latter may be hemangioblastomas. Striated muscle fibers were described in one (Gowers, 1876) and immature fat cells in another (Schnieder and Peiper, 1929) intramedullary lipoma (designated lipoblastoma by Bailey and Bucy, 1931). Only 16 reported intradural lipomas could be found by Bucy and Gustafson (1938), to which they added one. Those of Baker and Adams (1938), Jabotinski (1939), and the 3 of the present series raise the total to 22. These intradural lipomas are almost always dorsal, subarachnoid, seem to arise from the leptomeninges, and commonly invade the spinal cord parenchyma. They occur at any level and may be very extensive, as in the cases of Wolbach and Millet (1913), Eckart (1935), and Baker and Adams (1938). The first in this series extended from the medulla to the conus medullaris and markedly invaded the cord, and the last extended from the upper cervical to the lower thoracic segments and was associated with lipomas at the base of the brain and in the choroid plexus.

Some investigators, as Wolbach and Millet (1913), believed all of these tumors to be congenital, while others followed Virchow in considering them to be hyperplastic growths from fat cells normally found in the pia, such as were demonstrated by Boestrom (1897) and others before him. That there is a congenital factor present in their inception, is evidenced by their common association with congenital abnormalities, such as the spina bifida and meningomyelocele in our case and the cleft palate, harelip, and absent kidney in the case of Wolbach and Millet. Other evidence includes the occurrence of multiple lipomas, the coexistence of spinal gliomas, and the association of a subcutaneous lipoma in the dorsal midline in the same metameres as an intraspinal lipoma (Stookey, 1927; Bucy and Gustafson, 1938). Whether these tumors arise by inclusion of lipoblastic elements in the leptomeninges, the normal embryological occurrence of lipoblasts in the pia-arachnoid and their abnormal persistence, or by conversion of other mesodermal elements or their precursors into fat cells, is unknown.

METASTATIC INTRASPINAL TUMORS

Of the 254 tumors in this series 67 are metastatic and are classified as follows:

Sarcoma	32
Carcinoma	28
Hypernephroma	2
Neuroblastoma (sympathicoblastoma)	1
Lymphoepithelioma	1
Malignant lymphogranuloma (Hodgkin's disease)	1
Chloroma (myeloid)	1
Leukosarcomatosis of Sternberg (lymphosarcoma + leukemia)	1

Sarcoma

The 32 sarcomas are of the following types:

Sarcoma

Lymphosarcoma	20
Osteosarcoma	5
Reticulum cell sarcoma	3
Sarcoma (not further characterized)	1
Spindle cell sarcoma	1
Giant cell sarcoma	1
Leiomyosarcoma	1

Lymphosarcoma. All but one of the lymphosarcomas are thoracic, chiefly midthoracic; one occurs in the cauda equina region (Fig. 185). They extend over two to five vertebral levels, and often extensions through the intervertebral foramina from thoracic or abdominal neoplasms can be traced. All but one are extradural, and that one spreads diffusely through the cerebrospinal leptomeninges. Vertebrae are invaded in at least six instances and in these the epidural masses are probably secondary to the bone involvement. Although the dura is invaded a number of times, the tumor never reaches the subdural space. Only five of these cases came to autopsy and in these there is

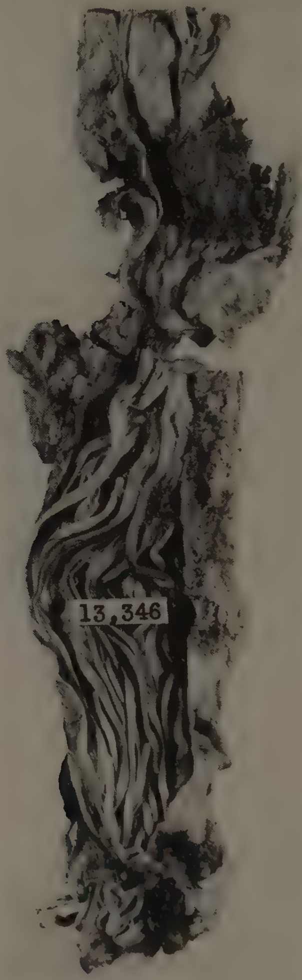


FIG. 185. Lymphosarcoma (T 11 to L 5). This is an extradural tumor compressing the dural sac, reducing the lumen of the spinal canal and compressing the roots of the cauda equina.

a generalized lymphosarcomatosis; in one instance, a lymphosarcoma of the stomach with metastases. The intranspinal neoplasms are reddish brown, pinkish, or yellowish gray; fleshy; firm and elastic, or soft and friable; and nodular or flat and sessile. Histologically, they are highly cellular, and the cells are small, closely approximated, and show no special arrangement (Fig. 186). Their cytoplasm is scanty, and they are rounded, oval or irregular. Their nuclei are spherical

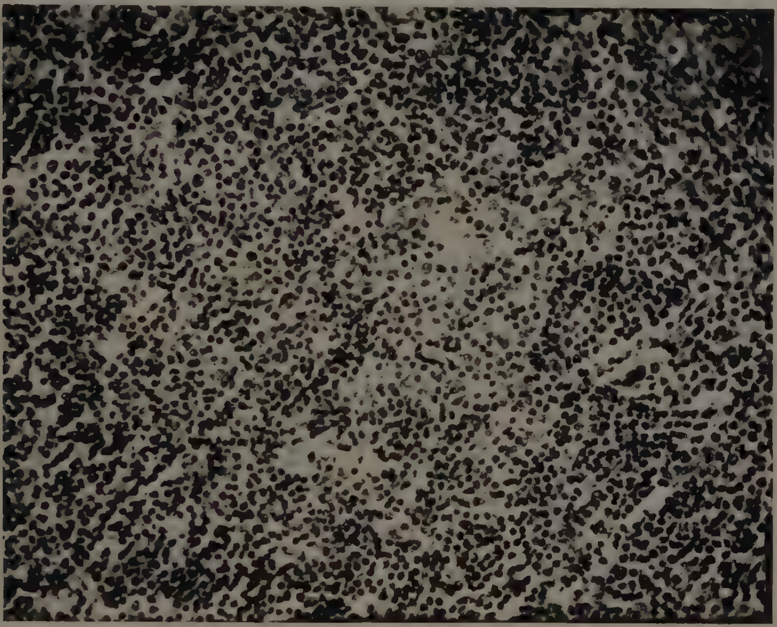


FIG. 186. Lymphosarcoma. The neoplasm was extradural from T 8 to T 10. It is extremely cellular. The cells are small and their cytoplasm is scanty. The nuclei are round, hyperchromatic and uniform. Hematoxylin eosin stain. ×110.

and hyperchromatic, and mitoses are frequent. They lie in a loose-meshed connective tissue. Davison and Michaels (1930) described four such tumors, none of which invaded the spinal cord, and Browder and de Veer (1939) eight.

Osteosarcoma. Four of these tumors arise from lower thoracic, lumbar or sacral vertebrae. They are extradural and elevate the dura, and in one instance invade the epidural fat and dura. They are reddish brown, irregular and soft, with some extremely hard areas. The cells are spindle-shaped, roughly rounded and polygonal, and the nuclei oval and hyperchromatic. Small and large giant cells with multiple hyperchromatic nuclei are encountered. Islands of osteoid tissue, cartilage, and irregular bone trabeculae are present in three and absent in one. There were multiple extradural metastases, cervical, thoracic and lumbar from an osteosarcoma of the left tibia. These are flat, granular and white, and resemble the first three just described.

Reticulum Cell Sarcoma. There are three of these neoplasms. One was epidural in the lower thoracic region, and another intradural extending from the conus down the cauda equina. These were surgical specimens, the first being large, soft, fleshy and vascular, and the second yellowish gray and friable. The third specimen was one obtained at autopsy; it invaded the subarachnoid space, spreading along the entire cord and along the nerve roots in the form of grayish plaques. In this instance, the primary tumor was retroperitoneal; it undoubtedly reached the spinal canal along the nerves passing through the intervertebral foramina. Histologically, these tumors are quite cellular, and the cells variable in shape and size. Many have abundant cytoplasm and some have large vesicular nuclei, although most of the nuclei are hyperchromatic. Multinucleated giant cells are fairly frequent and there is an abundant reticulum network. Necrosis occurs. One of the cases reported by Browder and de Veer (1939) was of this type.

Miscellaneous Sarcomas. The single *benign giant cell tumor*, removed from the fourth and fifth thoracic vertebrae, was extradural, dorsal, cystic, highly vascular, reddish brown, and rough and shaggy at the surface. The cystic core is soft and brownish. This tumor is quite cellular and vascular, the most prominent feature being the enormous giant cells containing a great many nuclei. Spindle cells and numerous proliferating capillaries are also present.

A retroperitoneal *leiomyosarcoma* had invaded the first three lumbar vertebrae and the epidural space, forming a lobulated, cream-colored, firm extradural mass which compressed the cauda equina and lowest cord segments. Histologically, parallel rows of very long spindle cells form interlacing bundles. Occasionally large multinucleated cells are present, and mitoses are numerous. A *spindle cell sarcoma* of the abdominal wall which had metastasized to the lower thoracic vertebrae, invaded the epidural space ventrally and compressed the cord. One *sarcoma of unknown origin* extended from the pleural cavity along the midthoracic nerves to the dura, nerve roots and pia of the midthoracic segments, resulting in a myelomalacia. This is a large cell sarcoma. Browder and de Veer (1939) reported an epidural *giant follicular lymphadenoma* of the upper thoracic region.

CARCINOMA

The 28 carcinomas were derived from the following organs:

CARCINOMA

Lung	11
Breast	4
Origin unknown	3
Prostate	2
Thyroid	2
Gall bladder	1
Colon	1
Testis	1
Parotid	1
Stomach	1
Arising in retroperitoneal teratoma	1

As in the intracranial cavity, lung tumors are the most common metastatic intraspinal carcinomas, the most numerous being the adenocarcinomas and oat cell tumors. The testicular tumor is an embryonal seminoma. The other tumors are typical examples of their various organ types, although there are often variations in morphology in the metastases, which renders identification difficult without autopsy, as in the three unidentified specimens. Direct extension or metastases to the vertebrae, or extension from thorax or abdomen by way of the intervertebral foramina, are the source of secondary epidural masses, which occur at all levels and compress nerve roots, cord, and cauda equina, producing varying degrees of degeneration (Fig. 188) (Keschner and Davison, 1933). All but 4 of the 28 metastatic carcinomas were purely extradural (Fig. 187). Three of the lung carcinoma metastases and 1 squamous carcinoma of unknown origin invaded the dura and presented on its internal surface; one of the pulmonary tumors invaded the leptomeninges and spinal cord. Such invasion is unusual. One lung tumor, whose gross intraspinal metastases were all epidural, gave rise to microscopic implantations in the spinal subarachnoid space, but diffuse carcinomatosis of the leptomeninges was not encountered in this series.

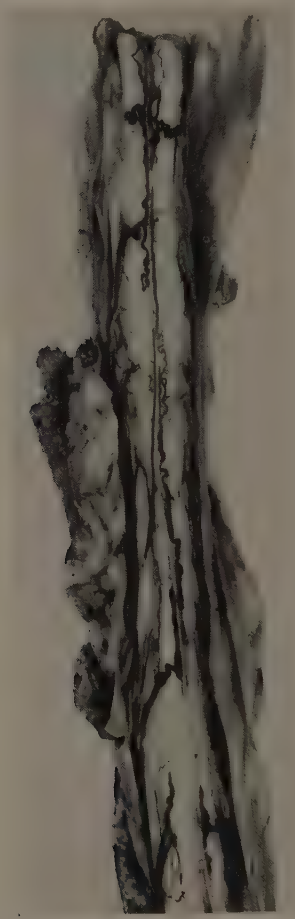


FIG. 187. A metastatic carcinoma invades the spinal epidural space and compresses the thoracic segments of the spinal cord (T 3 and T 4).

MISCELLANEOUS METASTATIC TUMORS

A *neuroblastoma* (sympathogonioma) of the adrenal, a *hypernephroma* of the kidney, and a *lymphoepithelioma* of unidentified source invaded vertebrae and epidural space and compressed the spinal cord. Each is typical of its kind.

MALIGNANT LYMPHOMAS

In *leucosarcomatosis of Sternberg* (leukemia + lymphosarcoma) a large mediastinal mass sent extensions through the intervertebral foramina to the upper thoracic epidural space, from which they spread into the cervical region; they were soft, white, and friable. This tumor extension looks much like a lymphosarcoma. There was one example of an intraspinal *malignant lymphogranuloma* (Hodgkin's disease) among the 253 intraspinal neoplasms of this series. It was extradural at vertebral levels T 5 to T 7, hard and white, and firmly attached to the dura and an intervertebral disc. The spinal cord was markedly compressed and showed intense myelomalacia. The growth is highly cellular, and contains lymphocytes, eosinophiles, plasma cells, polygonal cells with abundant cytoplasm, and large rounded giant cells with multilobed hyperchromatic nuclei lying in a highly fibrous connective tissue network.

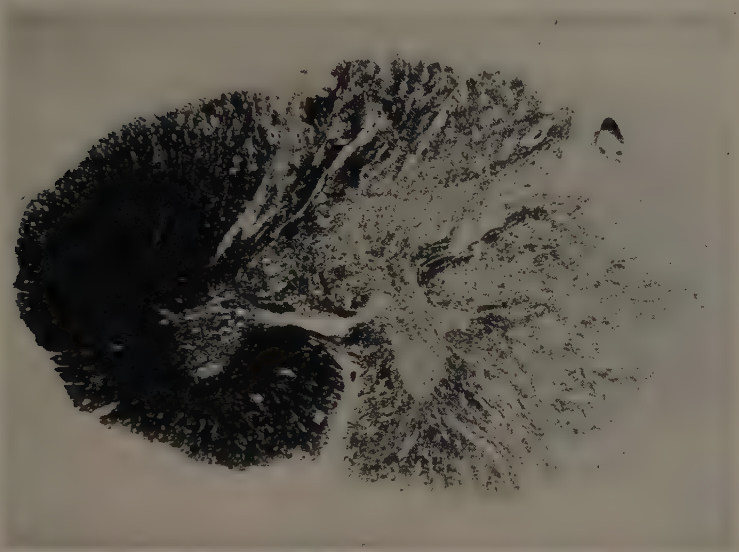


FIG. 188. Compression of thoracic spinal cord (T 4 to T 6) by a vertebral and epidural metastasis from a bronchial carcinoma. Much of the left half of the spinal cord is markedly degenerated. Pal Weigert stain.

Weil (1931) reported 3 cases of malignant lymphogranuloma with spinal cord involvement and collected 43 others from the literature. In 85 per cent there was an invasion of the spinal canal by epidural lymphogranulomatous tissue; in 7 per cent only epidural scar tissue remained following x-ray; and of the 4 remaining cases, 2 showed an infectious myelitis and 2 a syringomyelia. The dorsal segments of the spinal cord were involved in 80 per cent, the cervical region in 16 per cent, and the lumbosacral region alone in 4 per cent. Weil believed that mechanical obstruction of blood vessels passing through the lymphogranulomatous tissue, either within the intervertebral foramina or outside the spinal canal accounted for the myelomalacia observed in these cases.

A nodule of *myeloid chloroma* was present in the epidural space at T 10 in one case. It was large, firm, and decidedly green, and compressed the cord. In neither this instance nor in the leucosarcomatosis were there any additional intraspinal leukemic infiltrations.

Fried (1926) collected 30 instances of lesions in the central nervous system, associated with leukemia, and found 3 instances of intradural masses compressing the spinal cord (Eichhorst, 1898; Stursberg, 1914; Bassoe, 1918). Two of these were midthoracic and 1 lower thoracic and lumbar. They were reddish brown or grayish, flat and firmly adherent to the dura or vertebrae. Critchley and Greenfield (1930) reported 4 instances of intraspinal chloroma, 3 of which were extradural and 1 of which spread into the cord via the ventral fissure and the sheaths of the blood vessels. They reviewed the literature of neural involvement in leukemia, and noted (1) localized or diffuse leucocytic infiltration of the spinal cord (Rosenstein, 1881); (2) localized or widespread softening of the spinal cord due to occlusion of intramedullary vessels by white blood cells (Spitz, 1901); (3) a combination of (1) and (2) (Bloch and Hirshfeld, 1900); (4) hemorrhages into the spinal meninges (Olivier, 1877); (5) cellular infiltration of the meninges, chiefly epidural and dural (Eichhorst, 1898; Saltykow, 1909; Stursberg, 1914; Heissen, 1925) which was the most frequent complication and oftenest due to a chloroma; (6) infiltration of the lumbosacral plexus or peripheral nerves; and (7) the very rare cases of combined degeneration of the cord.

PRIMARY VERTEBRAL TUMORS

The vertebral angiomas have been discussed in the section on vascular tumors.

MYELOMA

In the present series, there are 4 myelomas (as a part of multiple myeloma) at various thoracic levels. Grossly they appear dark purplish brown, nodular and soft, or grayish white and firm. In 2 instances, the tumor extended into the epidural space and narrowed the spinal canal compressing the spinal cord; and in 2, deformation of the involved vertebrae caused angulation and similar compression. The tumors are highly cellular and the cells loosely arranged. They are oval, rounded or polygonal, and have abundant, homogeneous, slightly basophilic cytoplasm. Their nuclei are eccentric, spherical, and radially arranged. A clear perinuclear cytoplasmic zone is seen. Mitoses are frequent. In one tumor there are numerous lymphocytes, and there is some variation in the size and shape of the tumor cells.

As in most of the reported cases of vertebral myeloma (Denker and Brock, 1934; Davison and Balser, 1937) the above examples occurred in the thoracic region. Davison and Balser attributed the myelopathy in these cases to probable compression of the vessels of the spinal cord by the tumor, fractured vertebrae, or more rarely direct compression of the cord which seemed to be the cause in our examples.

CHONDROMA AND OSTEOCHONDROMA

There are three tumors of this type among the 253 intraspinal neoplasms of this series. Two are chondromas and 1 is an osteochondroma. Rasmussen, Kernohan and Adson (1940) reported 6 chondromas and 2 osteomas among 557 intraspinal tumors. One of our extradural neoplasms, a chondroma, was firmly attached to the fourth lumbar vertebra and the intervertebral disc between it and the fifth lumbar vertebra. It was right ventral in position, encapsulated, and cartilaginous in consistency. The second chondroma was left ventral, extradural, and extended from the fifth cervical to the fourth thoracic segment. It was a continuous mass of grayish white, glistening, cartilaginous tumor, and was attached to a number of vertebrae.

The osteochondroma extended from the fourth to the seventh cervical vertebrae. It involved laminae, pedicles, and spinous transverse processes and projected into the spinal canal. Its surface was bony and its interior spongy, cancellous bone contained fatty marrow and some cartilaginous zones.

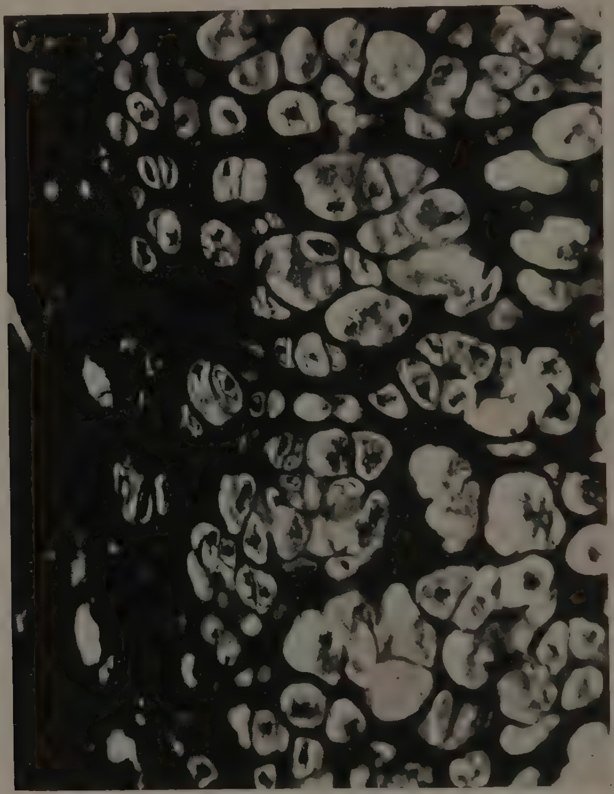


FIG. 189. Chondroma (C 5 to T 4). It is composed of abnormal appearing cartilage cells in closely approximated lacunae or clustered in a single lacuna. Hematoxylin eosin stain. ×230.

Histologically, the two chondromas are composed of hyaline cartilage containing considerable numbers of cells which vary a good deal in size and show no orderly arrangement (Fig. 189). The intercellular matrix is irregularly developed. The cells are rounded or stellate and have abundant eosinophilic cytoplasm which is often vacuolated and contains oval nuclei. The osteochondroma shows a similar structure in which partial calcification and ossification have occurred, particularly at the surface.

HERNIATED NUCLEUS PULPOSUS

Herniated nucleus pulposus ("ecchondrosis," "chondroma") and/or thickened and fibrosed ("hypertrophied") ligamentum flavum. In the present series of 346 intraspinal compressive lesions, 81 are instances of herniated nucleus pulposus of an intervertebral disc and/or thickened and fibrosed ligamentum flavum. In 33 cases there is herniation of the nucleus pulposus alone, in 27 it is combined with abnormal changes in the ligamentum flavum, and in 21 instances the ligamentum flavum alone is involved. The discs between L 4-5, L 5-S 1 and L 3-4 are most often implicated in that order, the first being by far the most frequent. The other instances are scattered among the lower cervical and lower thoracic intervertebral discs, among which the cervical abnormalities are more frequent, the disc between C 5 and C 6 being most often involved. For the combined lesion or the thickened ligamentum flavum alone, the distribution is almost exclusively lumbar.

It is generally agreed that trauma, single or repeated, acting upon a disc weakened by degeneration, produces the protrusion of the nucleus pulposus of the intervertebral disc (Schmorl, 1928; Andrae, 1929). Such disc degeneration, a phenomenon of ageing, is commonly present by middle age and may begin as early as the third decade or earlier, through the action of trauma or congenital defects in the supportive cartilage plates (Saunders and Inman, 1940) (Fig. 190). Andrae (1929) found cartilaginous nodules on the anterior surface of the spinal canal in 15 per cent of 368 spinal columns examined. These were most frequent in the fourth and fifth decades, and were almost constantly associated with evidence of disc degeneration. The process has been described by Saunders and Inman (1940) as an exaggeration of the normal tendency to desiccation of the disc with advancing age, occurring prematurely or to an excessive degree. There is, at first, nuclear expansion with central cavitation, and then softening due to a breakdown of cellular and fibrillar material. This is shortly followed by defects and erosions in the surrounding cartilage plates. The frequent "brown degeneration" is apparently the result of hemorrhage following secondary vascularization of the degenerating disc through the cartilage plates. The posterior position of the nucleus pulposus in the intervertebral disc explains its constant herniation in this direction. Rupture of the annulus fibrosus of the intervertebral disc with herniation of the nucleus pulposus or protrusion of the annulus alone or associated with such herniation may occur following trauma. Kubik (Mixter and Ayer, 1935) found nucleus pulposus and annulus fibrosus in most specimens removed, and this was confirmed by Saunders and Inman (1940).

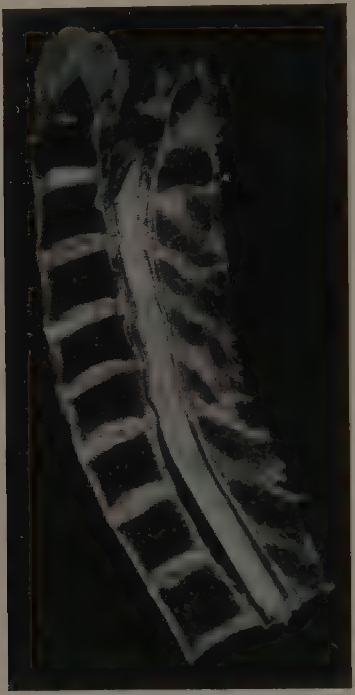


FIG. 190. This sagittal section of the cervical portion of the spine shows a fairly large protrusion and herniation of the fourth, fifth and sixth cervical discs with some indentation of the spinal cord. (Reproduced by courtesy of Saunders and Inman.)

Grossly, our specimens of herniated nucleus pulposus are gray or white, stringy, glistening, tenacious and soft or firm, silvery and cartilaginous. Histologically, they consist in most cases of incompletely formed cartilage, usually fibrocartilage which is evidently the result of cartilaginous metaplasia of the nucleus pulposus (Fig. 191). Cells are relatively infrequent and chondrification incomplete, the fibrillary ground substance often being quite prominent (Fig. 191). The cells are small, irregular, and shrunken or bipolar, and their nuclei are hyperchromatic. No islands of physaliphorous cells embedded in a delicate connective framework (such as are described under chordoma), which are present in the normal nucleus pulposus, were seen in any of our specimens. Occasionally fibroblasts, capillaries, and a few lymphocytes are seen invading this tissue. At its margins irregular strips of dense fibrous tissue composed of coarse collagenous bands, probably representing annulus fibrosus, are frequently found.

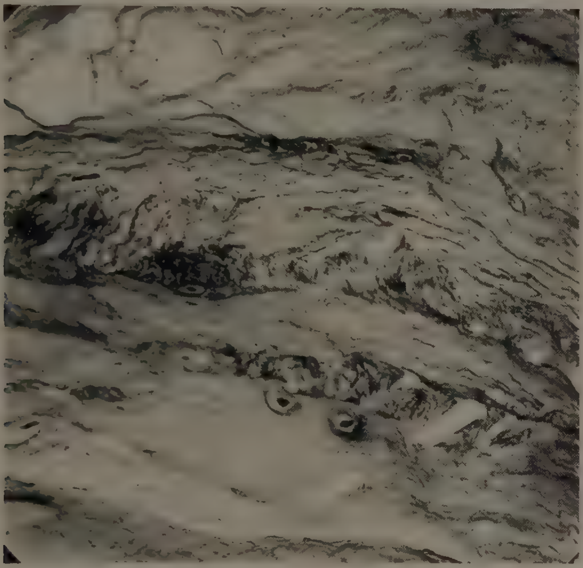


FIG. 191. Herniated nucleus pulposus between C 5 and C 6. There is a metaplasia to fibrocartilage. The cells are sparse and the fibers prominent. Chondrification is incomplete. There are no physaliphorus elements remaining. Hematoxylin eosin stain. ×300.

The thickened ligamentum flavum which is associated with such herniation in 27 instances and occurs alone in 21 shows a varying degree of transformation from the normal. Grossly it is yellowish or yellowish gray, firm and often inelastic. Histologically, focal areas of fragmentation and disappearance of elastic fibers (Fig. 192), with an irregular increase of fibrous tissue (Fig. 192) in these zones is found to a varying degree. Fibrosis and hyalinization of the walls of blood vessels is also seen. These changes involve almost half the tissue in somewhat less than half of the specimens examined, the rest being chiefly more severe and a small group less so.

Dockerty and Love (1940) examined 50 abnormal ligamenta flava and

25 normal controls from persons of all ages. No abnormal changes were encountered in the normal controls, while the majority of the others showed severe changes, consisting of splitting, fraying and fibrillation of the elastic fibers which "lost their specific staining characteristics and had taken on the appearance of connective tissue." This change was referred to as "fibrosis" and varied from minimal to almost complete replacement of the normal elastic tissue. In addition to the vascular changes just referred to in which some vessels were described as being occluded, in about one-third of the cases the number of blood vessels was increased. Three specimens showed calcification and two islands of chondrification. Lewy and Groff (1940) found 3 types of changes: (1) rupture of the elastic fibrils with fibrosis, (2) diffuse perivascular degeneration of elastic tissue, thought possibly to be due to a preceding inflammatory process, and (3) a hypertrophied ligament which was described as showing mild edema, swelling of the elastic fibers and thickening of the interstitial connective tissue. It would seem that the last was no true hypertrophy but rather one stage of a degenerative process with resulting fibrosis. This should be spoken of as thickening of the ligament as suggested by Dockerty and Love.

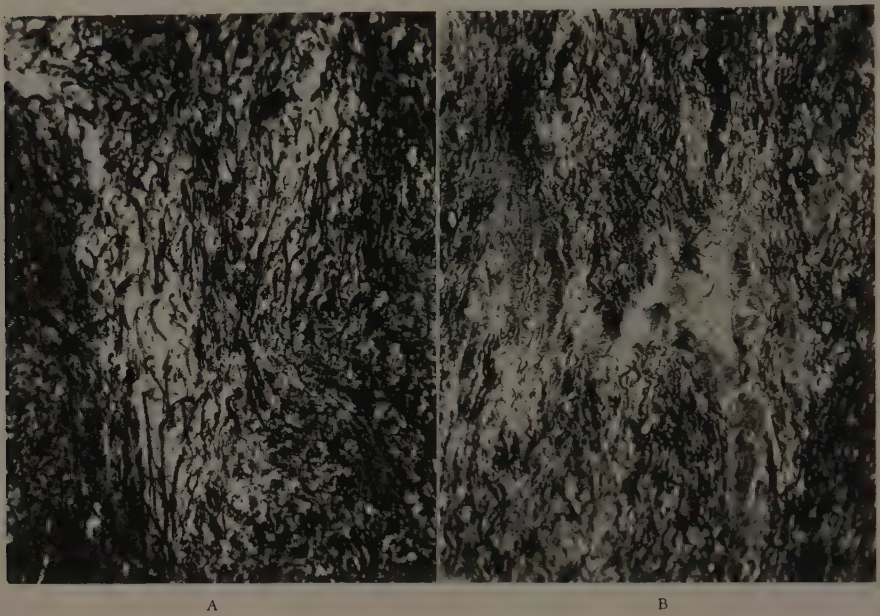


FIG. 192. Ligamentum flavum. Note the focal degeneration of elastic fibers and the accompanying fibrosis. Elastic tissue stain.

CHAPTER IX

TUMORS OF THE SPINAL CORD, NERVE ROOTS, AND MEMBRANES

III. SYMPTOMATOLOGY

SUBJECTIVE SYMPTOMS IN THE COURSE OF THE DISEASE

IN COMPRESSION OF THE SPINAL CORD BY TUMORS, THE ONSET OF SYMPTOMS IS A GRADUAL ONE, AND FROM THE BEGINNING, THE affection runs a chronic and progressive course.

In some patients, however, the onset may be acute. This occurred, for example, in one of my patients in whom the first symptoms appeared while the patient was walking, and consisted of a sudden loss of power in the lower limbs.

Cases of this kind are infrequent, but have been occasionally observed. Flatau (1911) reported a case in which, after a short illness, the lower limbs suddenly became paralyzed. Bregman and Steinhard (1907) mention a patient who was in perfect health until a paraplegia occurred which became complete in forty-eight hours; and Foerster (1901) speaks of one of his patients in whom the paralysis of the lower limbs became complete in one day.

The first symptoms of which the patients complain are usually sensory, as is evident from Table X, compiled from the case records of some of my patients:

In 20 of 275 patients, however, there were no sensory symptoms at the onset. In all but 3 of these patients, subjective sensory disturbances appeared sooner or later in the progress of the disease.

That an individual may have a new growth which makes increasing pressure on his spinal cord without causing any *pain*, is now well known. The absence of pain as an early symptom was formerly supposed to mean that the patient had an intramedullary and not an

Table X

SENSORY SYMPTOMS AT ONSET

Symptoms	*Extra-medullary*	*Extra-dural*	*Conus and Cauda*	*Intra-medullary*
Root pains	19	3	0	2
Pain in back or in neck	6	4	1	5
Pain in back extending down limbs	1	2	7	0
Pain in homolateral limb below level	1	1	0	0
Pain in contralateral limb below level	2	1	0	0
Pain in both legs below level	2	0	0	0
Pain in rectum	1	0	1	0
Pain in chest and abdomen	2	2	0	1
Tingling, burning, heaviness, pin-and-needle sensation or numbness in homolateral limb below level	1	0	0	4
Tingling, burning, heaviness, pin-and-needle sensation or numbness in contralateral limb below level	5	0	0	0
Tingling or numbness in both lower limbs	3	0	0	2
Tingling without pain, on same side	1	0	0	0
Tingling without pain, on opposite side	2	0	0	0
Numbness or heaviness but no pain	1	1	0	0
Feeling of stiffness but no pain	1	0	0	0
No sensory symptoms	1	1	2	2

extramedullary growth, until it was shown (Schultze, 1903, 1912; Stertz, 1906; Oppenheim, 1907, 1916; Nonne, 1908; Bailey, 1914; Serko, 1914; and others) that tumors may run a painless course. Serko stated that in one-half of the cases of extramedullary tumor that he collected from the literature, the patient had no pain, and that 29.5 per cent of the extradural growths ran a painless course.

My statistics are at variance with Serko's conclusions, for in my experience, *absence of pain* during the entire course of the disease is unusual. Nineteen of my patients declared that they never had had any pain; 8 had extramedullary and 11 had intramedullary tumors.

Writers on the subject of the symptoms of cord tumors have become accustomed to speak of a stage of root irritation and root pain, and a stage of cord compression and cord symptoms. While this is correct for those patients who do have root pain, it cannot be made to apply to the many patients who never have any real root pain. It is better, therefore, to speak of a *first stage, in which the symptoms are usually irritative in character*, and a *second stage, in which symptoms of interference with cord function become manifest.*

In the *first stage* must be placed all the symptoms which are complained of in the beginning of the disease, whether they be root pains, pain referred to some other part of the body, pain in the back, paresthesias, or other abnormal sensations.

Root pains are most frequent in intradural extramedullary tumors and occur in two-thirds of the patients with extramedullary growths, in about one-third of the patients with extradural growths, and in less than one-sixth of the patients with growths within the substance of the cord. The pain is usually of a neuralgic type referred to a part of the chest, of the abdomen or of the upper or lower extremities. In high cervical tumors the pain may be felt in one or the other suboccipital region or in the neck on one or the other side. A root pain at the fourth cervical segment is usually referred to the side of the neck or the top of the shoulder; a root pain of the fifth cervical root, to the outer aspect of the shoulder; while irritation of the sixth cervical root causes pain which radiates down the outer aspect of the upper extremity. Involvement of the sixth and seventh cervical roots causes pain which often begins in the thumb and index finger, while a root pain which begins in the fourth and fifth fingers is caused by an irritation especially of the eighth cervical sensory root. The pain from an affection of any of the cervical roots may be so severe that the head and neck are held absolutely rigid, and the question of actual involvement of the bony structures must be considered.

Pain due to involvement of the upper thoracic sensory roots is often referred to the axilla, under the scapula, or to an intercostal space, while lower thoracic root pain is often referred to the abdomen. The patients are often treated for intercostal neuralgia or for intra-abdominal disease. If the pain is in the upper abdomen, it may simulate pain due to disease of the gallbladder, duodenum, stomach, or kidney; and if referred to the lower quadrant (irritation of T 10 to 12) may be mistaken for an affection of the appendix, sigmoid flexure, or one of the pelvic viscera.

When the lumbar or sacral posterior roots are involved, the pain may be felt in the bladder or rectum, or it may involve a part of a lower extremity. When the pain is referred to the back of the thigh and to the outer aspect of the leg, it is often believed to be ordinary sciatica.

Root pains are often the earliest symptom of spinal cord tumor; they may appear suddenly, persist for months and then disappear as the symptoms of cord compression become manifest, or they may persist throughout the disease. There may, on the other hand, be intervals of freedom from pain.

In one of my patients with a tumor of the conus and cauda equina, root pains existed for fourteen years, and in another for nine years. Schultze had a patient who had localized root pain for sixteen years; and another in whom the root pain lasted for seven years, excepting a two-year period during which the patient was without pain.

In some of my patients, a root pain lasted for a number of months or for several years and then disappeared for a longer or shorter interval. Thus, one patient had pain in the back for six years and a free interval of two years before the pain reappeared; in others, there was a period of freedom from root pain of two years; twenty-nine months, and six years. Three of Antoni's patients had remissions in their symptoms, in one case lasting for ten years.

There may be not only remissions in the pain, but there are very often marked variations in the severity of the pain. The explanation of these remissions and variations is a simple one. Variations in the severity of a root pain may be due to a varying amount of edema and swelling of the tumor. After a tumor has reached a certain size, it may slip away from under a sensory spinal root with immediate disappearance of the pain. The remission will cease, however, as soon as the tumor has sufficiently increased in size to make pressure again upon the nerve root.

Root pains are always severe, and are often made worse by movement of the affected portion of the spine, and by forced expiratory efforts, such as coughing or sneezing.

Root pains may occur as an early symptom, not only in extramedullary, but also in intramedullary tumors, and may be the first symptom of a growth within the substance of the cord. Karger (1916) reported 2 cases of this kind, one a tuberculoma in the sixth to eighth cervical segments with pain in the left hand and arm, the other, a glioma in the ninth to eleventh thoracic segments with pain in the left hip. In both cases the meninges were normal. Intramedullary tumors, by enlarging in their transverse diameter, may cause root pains by pressing on the origin of the nerve roots from within outward, or through the pressure upon a sensory root of an irregularly swollen cord, or by the tumor itself after it has burst through the surface of the cord.

Root pain may appear late in the course of the disease when the growth has attained sufficient size to impinge upon a sensory root. Such a condition is met with in tumors which originate on the ventral or ventrolateral aspect of the cord or dura and grow backward; and in tumors which, though located on the dorsal aspect of the spinal cord, have begun in the space between two sensory roots.

The relief of root pain by changes in posture may be observed in tumors in every part of the spinal cord. In cervical cord tumors, the patient often holds his head and neck semiflexed and a little bent to one or the other side. In tumors of the thoracic segments, the bony spine is often held rigidly fixed in the most comfortable posture.

Pain may be due to involvement of the posterior gray horns or of afferent fiber tracts within the substance of the cord (Schultze, 1902; Antoni, 1920; Foerster, 1921, 1927; Elsberg, 1925; and many others) and is often projected into the periphery.

In a much larger number of cases than has hitherto been believed, the patients state that the first symptom they complained of was *pain in a distant part of the body over an area not supplied with sensation by nerve roots at the level of the tumor.* An onset with pain in one or both lower extremities is not at all rare in cervical and thoracic growths. Similar cases have been referred to by others (Ransom and Thompson, 1894; Oppenheim, 1907; Heilbronner, 1908; Flatau, 1911; and others).

Flatau's patient had an extramedullary tumor at the eighth cervical and first thoracic segments, and the first sensory complaint was pain in the left lower extremity. The case reported by Ransom and Thompson had initial pain in the legs although the tumor lay on the posterior surface of the eighth and ninth thoracic segments. The cases of Oppenheim and of Heilbronner were in this respect almost counterparts of the case of Ransom and Thompson.

The pain is not radicular in character or distribution, and is often felt in the lower limb of the opposite side. It must be an irritative phenomenon due to early involvement of the sensory pathways for pain in the spinal cord itself.

Pain in the back is a frequent early symptom of spinal cord tumors in every part of the spinal cord, but it occurs especially often in the lower part of the back in tumors of the lumbosacral cord and cauda equina. This pain in the back may be the only symptom for years. It is especially apt to occur when the tumor is adherent to the dura, and is, of course, very frequent in extradural malignant disease.

Pain in the small of the back is frequently made worse by sneezing and by coughing, and especially by changes in posture. Some patients will take peculiar positions in order to relieve the pain. Thus, in a patient with a tumor between the roots of the cauda equina, the pain was so severe when the patient was in the recumbent position that, for a long period, she could only obtain a modicum of relief day and night by sitting on a chair. In a recent case, the patient spent the greater part of the day kneeling with the body bent forward over the head piece of the bed.

Patients with a complete loss of cutaneous sensibility in the lower extremities, may suffer from pain when there are sudden reflex contractions of spastic lower limbs. I have heard patients complain bitterly of pain even when all the superficial and deep sensibilities had been lost.

Paresthesias. Tingling, burning, numbness, sensation of cold, "pin-and-needle sensation," and similar sensory disturbances are often complained of; and I believe insufficient attention has been paid to such paresthesias as an early symptom of cord tumors.

These paresthesias may be the result of slight irritation of one or more sensory roots by a tumor at the level of the roots, or may occur many segments above the actual level of the growth, and then be caused by the pressure of a column of fluid above the tumor.

It is possible that, as was claimed by Barré and Schrapf, these paresthesias above the level of the tumor may be due to sympathetic involvement. According to Langley, the fourth to tenth posterior spinal roots contain the greater part of the sympathetic fibers for the upper limbs, and tingling in the fingers (especially in the fourth and fifth fingers) may occur in tumors of the middle thoracic cord.

Paresthesias below the level of the lesion, undoubtedly the result of irritation of sensory pathways, are very frequently complained of.

These paresthesias were formerly supposed to be characteristic of disease within the substance of the cord, but in my experience, they are present in not a few patients with extramedullary new growths. While tingling in a limb is very often a sign of an intramedullary lesion, it is certainly not pathognomonic of disease within as contrasted with disease outside of the cord.

The location of these paresthesias is of considerable clinical importance. In a large number, in fact in the majority of instances, the patients stated definitely that the burning, tingling, or pin-and-needle sensation, when it appeared, occurred first on one side, the side opposite to that upon which the tumor was found at the operation. In many patients with extramedullary ventrolateral tumors, the first subjective sensory complaint was a tingling or burning in the lower limb of the side opposite to that of the growth. It is of interest that these contralateral paresthesias are rare in extradural tumors, although in one of my patients with an extradural tumor at the fourth and fifth thoracic segments, several toes of the contralateral foot had been amputated in another hospital on account of a burning sensation in the toes.

A certain number of patients have, either early or in later stages of their disease, a sensation of coldness in one or several extremities. The sensation of cold may be so extreme that it causes actual pain and prevents sleep. In one of my patients the sensation of coldness would come on very suddenly at night and would wake the patient out of a sound sleep. These sensations of extreme cold occur most often in tumors of the cervical cord.

Girdle sensations do not occur as frequently as was once supposed, and the location of the sensation does not always indicate the general level of the cord compression. A girdle sensation around the chest is not infrequent in cervical and high thoracic growths, but patients with a tumor in these parts of the cord more often have a girdle sensation in one or several extremities.

Subjective numbness occurs either as a root symptom, or as a result of the cord lesion. It is natural that the numbness complained of is on the same side as the tumor if it is a root symptom, or on the opposite side if it is due to interference with the sensory pathways of the cord for cutaneous sensibility. This subjective numbness may be complained of if either tactile or pain and temperature, or even articular or vibratory sensibility alone, has been diminished or lost.

The numbness which is due to the spinal lesion usually first affects the contralateral and then the homolateral limb; it is usually progressive and most often advances from the distal toward the central end of the affected limbs. It is quite rare for a patient with an extramedullary tumor to complain of a sensation of numbness which begins in the proximal part of the limb and extends into the most distal portions. In intramedullary tumors, on the other hand, the numbness often extends from the center toward the periphery.

In many instances, the diminution in sensation is first recognized by the individual when he discovers that he cannot distinguish between different degrees of temperature, and finds that hot or cold water is recognized less well over some portion of the body. This may be the only change that has been noticed by the patient, although the examination reveals disturbances of a cutaneous sensibility of touch, pain, and temperature.

A sense of weight or heaviness in the limbs is a frequent complaint, and a sense of weight in the abdomen is not rare in tumors at the level of the lower thoracic segments.

In a comparatively small number of cases, the first symptom noted by the patient is a *motor disturbance.* One patient with an extramedullary tumor in the lower cervical region, had a sudden loss of power in the lower limbs while walking so that she fell to the ground. One patient with a cervical extramedullary growth first noticed that he was awkward and stumbled when walking. One patient with a conus tumor first noticed a weakness in his lower limbs, and another with an extradural growth first noticed that he staggered and was unsteady. Three patients with intramedullary tumors observed, in the very beginning of their illness, a weakness of one or more of the extremities.

In all the other patients, motor symptoms appeared after sensory ones. When the tumor was located in the lower cervical region, the homolateral upper extremity was first affected, usually followed after a shorter or longer period by increasing loss of power and stiffness in the lower limb of the same side, later by an involvement of the lower limb of the opposite side, and finally by weakness in the upper limb of the opposite side. This order of involvement—(1) upper limb, (2) lower limb, (3) lower limb of opposite side and (4) upper limb of opposite side—was very frequent.

In thoracic cord tumors (below T 1) the limb on the side of the tumor is the one that first becomes weak, and the other limb becomes affected after a shorter or longer period of time. Not a few patients with cord tumors state that both lower limbs became weak and stiff at the same time.

Sometimes the stiffness is mentioned as having occurred first, but more often, the weakness was noticed by the patient before the stiffness. Some of the patients are unable to make reliable statements regarding the order in which the limbs were affected, and the statements regarding the bilaterality of the early motor symptoms must be accepted with some reserve. I have been unable to substantiate Antoni's claim that an interval between the motor involvement of the two sides of the body occurs in only one-half the patients with posterior growths (which Antoni claims are usually "neurinomas"), while in lateral growths ("endotheliomas") there is an appreciable interval between the involvement of the two lower extremities in every instance.

An exception to the rule that the motor symptoms first appear on the same side as the tumor (and also that the sensory symptoms appear first on the opposite side) occurs in those interesting cases of extradural or intradural growths adherent to the dura to which I called attention some years ago. In these cases the pressure effects of the tumor are altered and the motor disturbances first occur on the opposite side, and the sensory symptoms on the same side.

As has already been mentioned for subjective sensory symptoms, there may be considerable variation in the motor disturbances observed by the patient. In the majority of cases, the motor disturbances are steadily progressive and the patients declare that the weakness and stiffness of the affected limbs become constantly worse. In a few instances, however, there are periods during which weakness and stiffness are less marked. This has also been recorded by others. Thus, Babinski, Enriquez and Jumentié (1914) described a case in which the patient had a paralysis of the left leg which lasted for from two to three weeks, then disappeared entirely, and recurred several times after intervals of four months. Fuchs (1920) refers to a case in which the patient three times had a spastic paraplegia which cleared up entirely. The patient finally died, and at the postmortem examination an extramedullary tumor was found. The main features of the interesting case of Jumentié (1921) are contained in the title of his report: "Quadriplégie progressive avec rémission spontanée et guérison de trois ans suivies de rechute ayant entraîné la mort. Tumeur de la région cervicale de la moelle." These cases do not exhaust those that are to be found in the literature.

In extramedullary tumors the diminution in motor power and stiffness observed by the patients often begin in the most peripheral parts of the lower limb and progressively involve parts more centrally located.

The period of time which elapses between the beginning symptom and the first motor disturbance noticed by the patient, is, of course, subject to great variation, and is dependent upon the relation of the tumor to the cord and to the rapidity of the tumor growth. The period is shorter in intramedullary than in extramedullary tumors and is especially long in many tumors of the conus and cauda equina. There is the same variation in the time that elapses between the onset of the disease and complete paralysis of the lower limbs—if the cord compression has reached that advanced stage. Some patients with growths in the cervical or thoracic cord are seen at the end of a year with a complete spastic paraplegia, while in others the paralysis is incomplete after two or even three years.

As has already been mentioned, the onset of the loss of motor power is usually gradual, although, exceptionally, the onset of paralysis is very acute and the progress very rapid. An acute and rapid onset of paralysis is, however, much more characteristic of malignant extradural disease.

Involuntary flexor contractions of the lower limbs, often painful in character and, in advanced cord compression, accompanied by involuntary urination, are often complained of by patients. They occur relatively late in compression of the cord, at a time when spasticity of the limbs is already marked. These contractions may be slow and are mass reflexes evoked by a minimal stimulus, or they are quick and repeated—real clonic spasms—which are designated by the term spinal epilepsy, or by the French *trépidation epileptoïde.*

The story of the onset and progress of their disease as described by the patients varies with the location of the tumor and with the different relations of the growth to the spinal cord.

The patients may be seen in an early or late stage of their disease, but several types of history are often obtained:

1. A history beginning with neuralgic pain in an extremity or in the chest or abdomen for a number of months or years, followed, after a shorter or longer period, by motor disturbances on the same side of the body and sensory disturbances on the opposite side. The symptoms progress until both sides of the body are affected, and there is more or less loss of power and sensation on both sides of the body with spasticity of the lower limbs, and finally disturbances in the functions of the bladder and rectum.

2. A history beginning with tingling, numbness, "pin-and-needle" sensation or other paresthesia on one side of the body, followed after a period by progressive loss of power and sensation in the contralateral and later in the homolateral extremities. Finally, bilateral

motor and sensory disturbances and bladder and bowel derangements.

3. A history (often of long duration) of pain in some part of the back finally followed by motor and sensory and bladder and rectal disturbances.

4. A history beginning with increasing loss of power in one or both lower limbs, followed by subjective disturbances in sensation, and finally by bladder and rectal troubles.

There are, of course, many variations from these four types of clinical histories, as regards the manner of onset, the order of appearance of symptoms and the progress of the disturbances, that are observed by the patients themselves. Many a typical history of the onset and advance of symptoms of cord compression by a neoplastic process will be found to contain some unusual features, but the story of their disease as it is related by the patients often conforms in the main facts to one of the four groups just mentioned.

The most frequent atypical histories obtained are the following:

1. An acute onset with motor symptoms and, after a varying period, ill-defined sensory symptoms involving areas on both sides of the body. After a period, marked motor and sensory and bladder and rectal disturbances.

2. Symptoms referable to some abdominal or pelvic organ—stomach, gallbladder, appendix, bladder, rectum—persisting for years before motor and sensory disturbances appear.

3. A gradual onset with or without root pain or sensory disturbances followed by motor symptoms on the side opposite to the growth, and sensory symptoms on the same side as the growth.

4. Severe and persistent pain in one extremity with weakness of the extremity, but without other symptoms.

OBJECTIVE MOTOR, REFLEX, AND TROPHIC DISTURBANCES*

1. *During the first stage*, the only motor symptoms that occur are due to involvement of anterior spinal roots in extramedullary tumors

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* Involvement of special reflexes and of individual muscles is considered in more detail in Chapter x.

and irritative phenomena of the anterior horn cells in intramedullary growths. Theoretically, the first effect of irritation of a motor root should evidence itself by an increase of tone of the muscle supplied by the root. Patients do not, however, come under observation at this stage, but are first seen when the root has become involved to such an extent that the muscles in part supplied by it begin to lose power. When this occurs, the muscles will be found to be weak, and the tendon reflexes diminished.

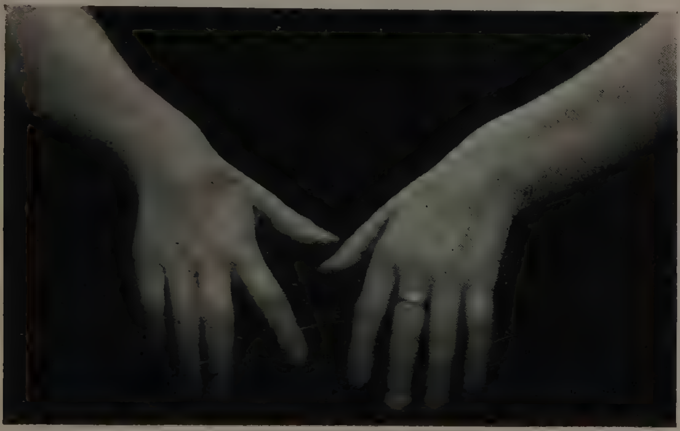

FIG. 193. Atrophy of the intrinsic muscles of the right hand in a patient with cervical spinal cord tumor.

As a motor-root symptom, weakness of part of an upper or lower extremity is often found at a period when there are as yet no spinal cord symptoms. Weakness or loss of a tendon or cutaneous reflex may, even at this period, have a localizing significance. Thus the early diminution in the power to extend one forearm at the elbow, or weakness or loss of the triceps reflex, may mean that the sixth or seventh and possibly the eighth cervical anterior root are involved, and the loss of one abdominal reflex may mean that the tumor lies at the eighth and ninth or the tenth and eleventh cord segments.

After a varying period of time, in pressure upon an anterior spinal root, the muscle itself begins to atrophy. The wasting is evident to the eye, as, for example, the atrophy of the intrinsic muscles of the hand that regularly occurs with involvement of the seventh (and eighth cervical and first thoracic) anterior roots (Fig. 193). These changes are, however, not alone found with anterior root involvement, as they occur also in affections of the cells in the anterior gray horns of the cord.

In intramedullary growths, the first motor disturbance is also in the homolateral limb, although in some instances, both limbs are affected at the same time.

As most of the muscles of the trunk and extremities have a plurisegmental representation in the cord, muscle power and cutaneous or tendon reflexes may be undisturbed if only one segment is affected. Thus, the cells in the anterior gray horns of the cord, which supply the axons for the motor innervation of the triceps muscles, are located in the sixth, seventh, and eighth cervical segments, but the power of the triceps and the triceps reflex may be very little diminished, when the lesion does not extend above the eighth cervical segment. This is, however, unusual, because a disease which involves one or several segments of the cord is generally accompanied by edema of the neighboring tissues and hence interference with the functions of more cord segments.

In the majority of instances, there is a diminution in the strength of the muscle and in the reflex of the tendon of that muscle. The muscle is more or less weak or entirely paralyzed, and the tendon reflex is much diminished or absent. In some cases, there is an inversion of the tendon reflex at the level of the lesion. I have seen such an inversion of the triceps reflex in patients with tumors at the seventh and eighth cervical segments. In this instance, the attempt to elicit a triceps reflex always caused a flexion of the forearm through contraction of the biceps and (especially) the supinator longus, instead of the normal extension movement. This inversion of the triceps reflex, is often seen in acute and severe cord lesions at this level, such as occur in fractures of the spine with crushing of the cord. I have, in some instances of high cervical tumor, observed the change in the radius reflex described by Babinski, in which percussion over the styloid process of the radius causes a flexion movement of the fingers.

Weakness and atrophy of muscles which are innervated through segments of the cord above the actual level of the growth are occasionally observed, and it is probable that in these patients the involvement of muscles above the lesion is due to an edema of the cord.

2. *Motor and Reflex Disturbances at and just below the Level of the Growth.* As soon as a tumor has begun to make pressure upon the cord itself, it will not only interfere with reflex centers in the affected segments, but will block efferent impulses which travel in the descending fiber tracts, with the result that motor and reflex disturbances, in parts supplied from cord segments below the level of the lesion, become manifest. The combination of weakness or loss of power in muscles, and disappearance of reflexes whose centers are in known segments of the cord, with changes in reflexes below this level, is of great diagnostic significance.

The motor and reflex level signs that I have most often met with in localized compression of the cord are: Absence of biceps with exaggeration of triceps reflex with weakness of flexion and extension at elbow, in tumors at the fifth to sixth cervical segments; normal biceps and weak or lost triceps reflex with good power of flexion and more or less diminished power of extension in tumors at C 7 and C 8; diminution or loss or triceps and ulnar reflexes with spinal miosis in lesion of C 8 and T 1; diminution or loss of upper and lower abdominal reflex on one or the other or both sides, as a level sign in tumors of T 8 to 9 (for the upper) and T 10 to 12 (for the lower abdominal reflex) ; diminution or absence of one or both cremasteric reflexes (L 1-2) with exaggeration of one or both knee jerks (L 2-4), with a tumor at the first lumbar segment; absence of one or both knee jerks, with ankle clonus on one or both sides, in tumors at the second to fourth lumbar segments. In the lumbosacral cord, the segments are small and close together, and it often happens that a tumor will involve most of the lumbar segments together with the entire sacrad cord, so that cremasteric reflexes, as well as knee and ankle jerks, are lost.

In one instance of tumor on the left side of the lumbosacral cord, we observed a remarkable ensemble of physical signs: on the left side flaccid paralysis of the limb with drop-foot, absent knee and ankle jerks; and on the opposite (right) side, spastic paralysis with exaggerated knee jerks, ankle clonus and Babinski. The tumor compressed the roots of the cauda equina on the one side, and the pyramidal fibers on the other.

The loss of power of muscles whose spinal centers lie in segments of the cord that are compressed by a tumor, is often, but not always, accompanied by a more or less well-marked atrophy of the affected muscles. Formerly such an atrophy was considered almost diagnostic of disease within the substance of the cord. It occurs also with extramedullary growths, and is then due to pressure transmitted to the anterior horns of the cord. Such muscle wasting at the level is more apt to occur in tumors on the ventral or ventrolateral aspects of the cord although it occurs also in posterior tumors when the compression is far advanced. In tumors which compress the upper cervical segments, atrophy of the muscles of the forearms and hands (below the level of the lesion) is quite frequent.

Fibrillary twitchings, while more frequent in intramedullary disease, are often observed in tumors outside the cord, in muscles supplied from segments compressed by the growth. These fibrillations will be found not only in wasted muscles, but also in those that show no evidence of atrophy; and the more carefully I have searched for this muscle fibrillation, the more often have I found it. Fibrillation of muscles of the upper extremities is fairly frequent in high cervical tumors, and fibrillation of the glutei in thoracic tumors. The fine fibrillary twitching of the muscles of the legs that is often seen in tumors of the lumbosacral cord and roots of the cauda equina, is not a cord sign, but a nerve-root sign in muscles that have undergone a degenerative atrophy.

3. Motor and Reflex Disturbances below the Level of the Growth. In the incomplete isolation of the cord in compression by tumor, the pyramidal pathways are gradually interfered with, and to a greater or lesser extent the final common pathway is deprived of the influences which work upon it by way of the upper motor neurons. When the pyramidal pathways are completely or partially interrupted, there is a diminution or loss of voluntary power and an increase of the tone of the muscles from the unrestrained activity of the spinal motor centers.

Spasticity and hypertonus, the exaggeration of the tendon reflexes, and the Babinski or extensor type of response of the large toe (upon plantar irritation) and its modifications, are therefore the expression of uncontrolled spinal reflex activity, and the extent of the disturbance is dependent upon the degree of the interference.

The amount of motor disturbance depends, therefore, upon the degree of compression.

Unless a tumor is situated in the midline on the ventral or dorsal aspect of the cord, the motor disturbances are observed first in the extremities of the side of the tumor, followed sooner or later by weakness (and spasticity) of the limbs on the opposite side. As the disease progresses, the weakness and spasticity become more and more marked, but the limb on the affected side is, in the majority of instances, weaker and more spastic.* Even when there is a complete paralysis of the lower limbs, the history of the development of the paralysis will indicate that one limb was affected before the other. Furthermore, even with limbs completely paralyzed and spastic, one limb may show more marked disturbances in the reflexes than that of the opposite side.

Both limbs may appear to be equally spastic, but the reflexes may not be elicitable in one extremity on account of the stiffness, while there are still exaggerated reflexes on the other side. This means that the limb in which reflexes cannot be obtained is the limb in which the motor disturbance is most advanced.

In extramedullary tumors the motor disturbances in the limbs usually occur in a regular order, but exceptions to this rule are fairly frequent. If the tumor is above or at the level of the motor roots for the upper limbs, the homolateral upper extremity is first involved. Unless a motor defect is due to anterior root or anterior gray-horn-cell involvement, the diminution in strength affects first the most peripheral part of the limb and gradually involves more and more of the extremity. Motor symptoms in the homolateral lower extremity next appear, followed by disturbances in the power of the contralateral lower and finally of the contralateral upper extremity.

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* Spasticity does not occur in tumors that compress the roots of the cauda equina.

The contralateral upper limb may be affected before any of the lower limbs, and more rarely, there is a loss of power in the contralateral upper extremity before the change in the homolateral lower and before any alteration in the contralateral lower extremity.

Sometimes both upper, and later both lower, limbs are involved together, but the changes are usually more marked in the limb on the same side as the spinal tumor.

If the growth lies below the cord level for the upper extremities, then, obviously, only the trunk and lower limbs are involved in the changes, and the homolateral precedes the contralateral limb.

In extramedullary tumors at any level of the cord, the motor disturbances in the lower extremities may begin in the most peripheral parts of the limbs, and weakness and spasticity spread up the limbs from the toes and feet. This never occurs in intramedullary growths, where the motor weakness spreads, just as does the sensory disturbance, from the center toward the periphery. In growths within the substance of the cord, contemporaneous motor loss in the two upper or in both lower extremities is more frequent.

In some instances the motor disturbance first occurs in the contralateral limb, and is due to a marked dislocation of the cord to the opposite side with pressure of the pyramidal tracts of that side against the wall of the spinal canal. This contralateral motor disturbance is often met with in extradural and intradural growths but it is also seen in intradural growths adherent to the dura. Nonne and Oppenheim have described cases of this kind without having given any explanation for them; and Antoni (1920) reports a case of "dural endothelioma" with a contralateral paresis of the arm and leg, but he, also, states that he is unable to give an explanation for the occurrence.

The amount of spasticity and the parts of the body involved vary with the location and degree of the compression of the cord. In high cervical tumors, one or both upper or lower extremities may be spastic, and the stiffness may involve only or mainly the limbs of one side. In tumors below the cervical level, the abdominal muscles are rarely, and the lower extremities are regularly, involved in the spasm. The spasticity of the upper extremities is most often a stiffness in flexion, while in the lower limbs, the spasticity may be in flexion or in extension. When the patient is at rest in the recumbent position, the limbs may be relaxed, but the smallest irritation causes strong flexor or extensor contractions of the lower limbs. One limb may be drawn up in flexion, while the other is in a state of extensor spasm. There are sometimes remarkable changes in the spasticity: at one examination, the lower limbs are found in marked adductor spasm, and on the following day, perhaps, all spasticity has disappeared. I have seen this in a number of instances; and similar cases have been reported by Fuchs (1920), Foerster (1921), and others.

The spasticity of the affected limbs may be so great that voluntary movement is impossible. On the other hand, the patient may be able to move the toes, and with great effort, partly to flex the leg at the knee.

The weakness of the affected limb or limbs is usually distinct, although the patient's statement that he has no power at all in the limb must not be taken as correct. When the possibility of movements at the various joints is tested, slight motor power may be found to be preserved in one or another joint.

The cutaneous reflexes are regularly diminished or lost and the tendon reflexes increased below the level of the compression by the tumor. Thus the abdominals and cremasterics are almost always lost in well-marked compression of the thoracic cord above the centers for those reflexes.

In high cervical compression (C 1-C 4) all the tendon reflexes on one or both sides may be exaggerated. The triceps reflexes are frequently exaggerated alone in tumors above the sixth cervical segment. It is a very interesting fact, to which Oppenheim first called attention, that the diaphragm may not be paralyzed, even with complete transverse symptoms above the fourth cervical segment.

The suprapatellar, patellar, and Achilles reflexes are regularly exaggerated and the Babinski reflex (extension of large toe) and many of its modifications elicitable. The knee and ankle jerks on the side of the tumor are usually more active than those of the opposite side and there is usually a transient or an inexhaustible ankle clonus. The ankle clonus may be inexhaustible on the side of the tumor and easily exhaustible on the opposite side.

The threshold of the reflexes is often markedly lowered so that a minimal stimulus will give rise, not only to the expected reflex, but to a large number of additional reflex contractions. These are the mass reflexes, so often seen in patients whose limbs are very spastic, about which much has been written by Walshe, and by Head and Riddoch.

While mass reflexes appear in their typical form in complete transverse lesions of the cord, they are also observed in incomplete spinal lesions from compression by tumor. Thus irritation of the sole of the foot may cause a movement of one or both lower limbs as well as a contraction of the abdominal muscles and reflex evacuation of the bladder.

Very often, scratching the sole of the foot will cause not only dorsal extension of the large toe, but "fanning" of the other toes with dorsal flexion at the ankle and flexion at the knee and hip. The *réflexes de défense* described by Babinski, are really mass reflexes evoked by a minimal stimulus.

There is considerable probability that the extension of the large toe which occurs upon plantar stimulation is also a defense movement. It is often associated with dorsal flexion of the foot; and both extension of the large toes and dorsal flexion at the ankle may be evoked by stimulation of the skin or the deeper structures above the foot itself.

The *signe de la flexion réflexe du pied* of Babinski, which consists of dorsal flexion of the foot obtained by irritation of the skin of the thigh or leg, is frequently seen in compression of the cord by new growths. It is probably a reflex similar to that described by Schaefer, and to the *Unterschenkelphänomen* of Oppenheim.

In many patients with the signs of cord compression, defense reactions of the lower limbs can be observed, and in tumors of the upper cervical segments, such defensive reflexes may occur in the upper limbs. The least irritation of the skin anywhere below the level of the lesion may cause powerful and often painful contractions of the limbs. The irritation of the bedclothes is often sufficient, and thus a satisfactory examination of sensation and of the tendon reflexes may be difficult.

These *réflexes cutanés de défense* of Babinski are often well marked in spinal-cord compression by tumors, but there does not seem to be any direct relation between the degree of the compression, the secondary degenerations and the intensity of the reflex movements. In some instances the defense reactions were especially severe, although the cord was only moderately pressed upon by a small tumor and the patient improved very quickly after the growth had been removed.

The defense reactions in the lower limbs are most frequently of the flexor type and are associated with an extensor movement of the large toe, although they may occur without the latter. The flexion movement at the ankle, knee and hip joints is usually sudden and of short duration, but the relaxation that follows occurs more slowly. The muscular contractions may involve also the muscles of the abdomen and chest and even those of the upper limbs, if the lesion be above the spinal centers for the upper extremities.

The *réflexes de défense*, called by a variety of names (mass reflexes, *réflexes d'automatisme médullaire, phénomène des raccourcisseurs,* etc.) have evoked a great deal of discussion as to their nature. Babinski believes that defensive reflexes occur only up to areas which correspond to the lowest cord segment involved by the growth, and that therefore the lower level of a growth can be diagnosed from the highest level of the reflex contractions. In a few instances of tumors of considerable length, I have been able to verify the correctness of Babinski's contention. Most spinal new growths are relatively small, and in these I have gained no information as to the size of the growth from the study of the defense reflexes.

In compression of the cord by new growths, both sides of the body are usually involved, although the reactions may be more intense on one side. Not so rarely, excitation of the skin of one lower extremity will be followed by a withdrawal movement of both lower limbs. We have only rarely observed the crossed extension reflex (Phillipson) in which there is an extension movement in the opposite limb and a flexion movement on the stimulated side.

The threshold of stimulation for a reflex may be so much lowered that the reflex is obtained by irritation of the skin far away from the usual site from which the reflex can be elicited. I have frequently seen the cremasteric reflex elicited, not only by scratching or pinching the skin of the thigh, but also the skin of the leg, the foot, and even the toes.

In acute cord compression, there may be complete flaccid paraplegia, with loss of all cutaneous and tendon reflexes as a sign of a complete transverse cord lesion. In the slow and gradual compression of the cord by new growths, this rarely occurs; the limbs, though completely paralyzed, remain spastic for a very long period.

This is quite different from what occurs in acute traumatic lesions, in extraspinal malignant disease with paralysis from interference with the vascular supply of the cord, and in the acute compression of the cord that occurs with sudden collapse of one or more vertebrae. In these latter states, there is a more or less prolonged period of flaccid paralysis.

In tumors of the lumbosacral cord and cauda equina the loss of reflexes is due to the pressure upon the nerves of the cauda equina rather than to the pressure upon the lower end of the spinal cord, and in these patients flaccid paralysis is the rule.

Ataxia and tremor of one or more extremities from pressure upon the spinocerebellar tracts on one side, is rarely observed, because the pressure usually involves the pyramidal tract at the same time. It is then impossible to distinguish an unsteadiness due to motor weakness from pure ataxia. In one instance, however, I examined a patient in an early stage of cord compression, who had ataxia of one lower extremity without motor weakness. He was later operated upon in another clinic, and a spinal cord tumor removed from the midthoracic cord on the side of the ataxic lower extremity.

Excluding high cervical growths in which wasting of the muscles of the upper limbs is frequent, atrophy of the muscles of limbs *below* the level of the lesion is rare, excepting with very advanced signs of cord compression. It is then an atrophy of disuse and a part of general bodily emaciation. In this advanced stage, edema of the legs and feet is frequently observed, as are also atrophic disturbances of various kinds.

Decubitus over the sacrum, buttocks, or heels rarely occurs in compression by tumor excepting in compression of the conus and cauda equina, where it is frequent, and in the advanced stages of cord compression with a lesion which involves almost all or all of the entire diameter of the cord. The more acute the cord lesion, the more apt are bedsores to occur.

Contractures occur sooner or later after paralysis and spasticity of spinal origin, just as they do after hemiplegia due to cerebral injury or disease. These contractures are rare in the upper extremities excepting when the tumor is at the level of the highest cervical segments or when the contracture has followed a long standing paralysis of an upper limb as a result of involvement of a number of anterior spinal roots. In the lower limbs, contractures are often seen after a long standing and sometimes irremediable cord lesion that has resulted from the pressure of a tumor.

Our experience with the *pilomotor reflexes*, concerning which valuable and interesting studies have recently been made by André Thomas (1921), is not sufficient for us to make definite statements as to the changes that occur in compression of the spinal cord by new growths.

André Thomas divides these reflexes into two groups: a spinal reflex which follows stimulation of the extremities, and an encephalic reflex obtained by pinching the muscles at the back of the neck. Under normal conditions, the impulses set up by the appropriate stimulation pass, respectively, upward and downward in the spinal cord. According to the author, the two reflexes are isolated from each other when the cord has been compressed sufficiently to cause automatic activity of the segments below the level of the compression. In such a case, the spinal reflex travels upward and the encephalic reflex downward to the affected level of the cord, but not further. Continued investigation must be relied upon to demonstrate the exact extent and value of these pilomotor reflexes.

Reflex sweating is sometimes observed in compression of the spinal cord, especially when the compression is far advanced. Head and Riddoch (1918) made careful studies of reflex sweating in patients with completely divided spinal cords, and showed that, under certain conditions and with certain stimulation, sweating can be produced over the face, neck, and upper extremity of one or the other side.

In some of the patients of our series, with cervical cord tumors, reflex sweating was observed; this occurred either without any stimulation, or followed various modes of excitation. The sweating was most often observed on one side of the face and neck, but we have seen reflex sweating strictly limited to one or two dermatomes. Thus, in one patient recently operated upon, with an intramedullary growth at the third and fourth cervical segments, there was profuse sweating over the third and fourth cervical dermatomes on both sides. In another patient with an extramedullary tumor to the right of the fifth cervical segment, the sweating involved the right side of the face and neck to the level of the clavicle.

In most patients, *disturbances in the functions of the bladder and rectum* occur relatively late in the progress of the disease, although sooner or later every patient with a compression of the cord will develop bladder and rectal symptoms. Even in tumors of the conus and cauda equina, bladder disturbances rarely appear early. In the patients of this series the late appearance of interference with these functions lends support to the view, expressed a number of years ago by Spiller, that bladder and rectal symptoms need not appear early in intradural pressure upon the roots of the cauda equina.

The mechanism of bladder control and the innervation of the bladder and its sphincters are not yet well understood. Little is known of their central paths in the brain and spinal cord. There is considerable evidence to support the view that in the spinal cord the fibers for the bladder run in a juxtagriseal pathway. According to Holmes and Walshe (1919) the sympathetic connector fibers leave the cord by the two lower thoracic and the two upper lumbar roots and enter the hypogastric nerve, while sacral fibers come from the second, third and fourth sacral segments and join the pelvic nerves. There is a third or voluntary element in the nervous mechanism of micturition which passes by the pudic nerves to the striated muscle of the urethra. The functions of the bladder are under the combined control of these three mechanisms, and this is the probable explanation why bladder symptoms may be slight when only one of them is involved.

In tumors above the lumbosacral cord, the first difficulty in urination is usually dysuria—a difficulty in starting the part of the expulsive act which is under voluntary control. This may lead to retention of urine until the intravesical tension and the automatic rhythmic contractions of the bladder musculature overcome the internal vesical sphincter and the urethral muscles, and, unless retention is relieved by catheterization, involuntary urination occurs. The overflow may be more or less continuous as the muscular mechanism of the bladder wall becomes more and more interfered with, so that the patient seems to be incontinent of urine.

True vesical incontinence probably rarely occurs excepting when all three mechanisms are interrupted by a growth over the lumbosacral cord, with a loss of reflex and voluntary power in the musculature of the bladder and all the sphincters.

In tumors, as in crushing injuries of the lumbosacral segments, there is true incontinence of urine with relaxation of the bladder musculature and all the sphincters. Even here, a certain amount of automatic bladder activity will often reappear.

It is exceptional for bladder disturbances to occur early, and early vesical incontinence is usually indicative of an intramedullary rather than an extramedullary growth in the lumbosacral cord or of a tumor of the filum terminale.

Constipation is almost the rule in spinal cord tumors, and incontinence of feces is rare, excepting when the intestinal contents reach the colon and rectum in a fluid state such as occurs after the taking of a laxative. The mechanism of defecation is not well understood, but the spinal centers probably lie in the same segments as the centers for the bladder, and are influenced in the same way as are the vesical centers.

OBJECTIVE SENSORY DISTURBANCES

1. *The First Stage.* In the early stages of the symptoms of a tumor of the cord, objective sensory disturbances are usually absent. If the growth has originated from, or has from the beginning been in close proximity to, a sensory root, careful examination may show that somewhere along the corresponding skin area there are patchy areas of the skin that are hypersensitive to pin-prick or to hot and cold.

Sooner or later, this hypersensitiveness becomes distinct over a small or large part of a root area. If several sensory roots are involved by the growth, a distinct diminution or loss in sensation will occur. There are many exceptions to the so-called Sherrington's law—that an area of cutaneous anesthesia will be found only when the afferent impulses through three successive spinal roots have been interfered with. Root anesthesias may occur when only two roots are involved but a localized hyperesthesia of the skin may be found where only one spinal root is implicated by the growth.

Radicular hyperesthesia or anesthesia may occur over any part of the body, dependent upon the location of the tumor. In growths at the upper cervical levels, root disturbances may be found in the third cervical dermatome (over the neck and posteriorly to above the occiput) or on the lower part of the cheek below the area of distribution of the inframaxillary part of the trigeminus. It has surprised us that, in spite of the fact that the spinal root of the fifth cranial nerve must have been involved in the pressure, there was no disturbance in any part of the trigeminal area in any of the high cervical growths.

In tumors of the conus and cauda equina, root hyperesthesias or anesthesias are often to be found in some parts of one or both lower extremities.

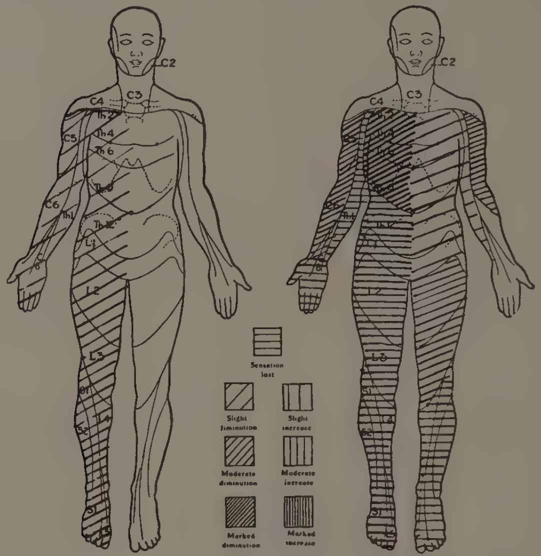
In intramedullary tumors, there may also be a root hyperesthesia when the growth, in this first stage, has begun in close contact with the zone of entrance of the roots. Diminution of cutaneous sensibility due to the local effect of a tumor within the substance of the cord is always, as in extramedullary growths, on the side of the maximum lesion, and on the same side as the loss of motor power and the muscular atrophy. In some of our patients, the sensory disturbance at the level of the growth showed definite signs of intramedullary segmentation.

The irritation by a neoplasm of fibers of the ascending pathways in the cord may result in hypersensitiveness of an area of skin far distant from the segmental level of the growth. A disturbance of sensation from this cause, however, belongs to the second stage of interference with cord function.

2. *The Second Stage.* In this stage the amount and extent of the cutaneous sensory disturbances will depend upon the amount of blocking and of actual destruction of cord fibers, and will vary with the location of the growth and with its relation to the cord.

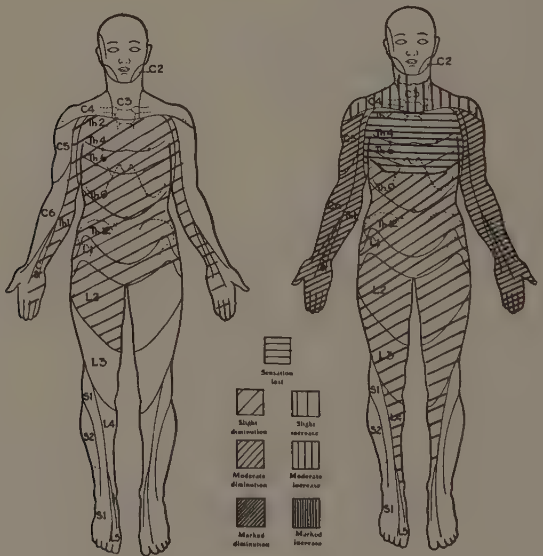
Unless all cutaneous sensibility has been lost, there is, in many extramedullary tumors below the level of the lesion, a certain amount of dissociation of sensations (Fig. 194). Insufficient attention has been devoted to this fact. I have seen more than one patient in whom intramedullary disease had been suspected because there was dissociation of cutaneous sensations, tactile being not all or only slightly affected, while pain and temperature were markedly diminished or lost.

In one patient cutaneous sensory disturbances occurred only after a lumbar puncture had been performed. During the period when the power to feel the pain of a pin-prick and to recognize and distinguish between hot and cold below the segmental level was becoming diminished until these sensations were abolished, light touch with cotton wool was felt distinctly and equally all over the body. There was therefore a period during which there was typical "sy-



Touch Pain and Temperature

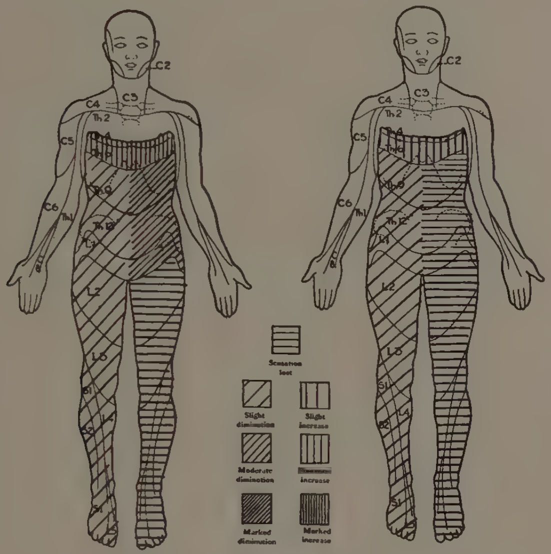
FIG. 194. Cutaneous sensory disturbances in a patient with an extramedullary cord tumor at the fifth cervical segment, showing marked dissociation between tactile and pain and temperature sensibility.



Touch Pain and Temperature

FIG. 195. Cutaneous sensory disturbances in a patient with an intramedullary tumor at the fifth cervical segment, showing dissociation between tactile and pain and temperature sensibility.

ringomyelic" dissociation in which tactile sensation over the trunk and both lower limbs was normal, while pain and temperature were profoundly altered. In another patient with a tumor at the level of the sixth, seventh and eighth thoracic segments, pain and temperature sensibility were lost over the lower half of the abdomen and both lower extremities, while tactile sensation was only slightly involved below the segmental level of the lesion.



Touch   Pain and Temperature

FIG. 196. Cutaneous sensory disturbances in a patient with a reversed Brown-Séquard syndrome from an extradural tumor at the fourth thoracic segment. The tumor lay on the left side but the most marked disturbances of motor power and reflexes were on the right side, and the most marked disturbances of cutaneous sensibility on the left.

In intramedullary tumors, on the other hand, all types of sensation were frequently equally involved, while typical dissociation—preservation of tactile with marked diminution or loss of pain and temperature sensibility—was rarely seen. The differences in the involvement of the three types of cutaneous sensibility were no greater than those often seen in extramedullary new growths (Fig. 195). It may be that the patients came under our observation in a late stage of their disease, but I am inclined to believe that in true expanding lesions within the substance of the spinal cord, dissociation of sensation (excepting in a very early stage of the disease) is not as frequent as in syringomyelia, hematomyelia, and spinal gliosis. This relative freedom from disturbance of tactile sensibility in many extramedullary as well as intramedullary tumors may be due to the fact that the pathways in the spinal cord for this variety of cutaneous sensibility run a bilateral course, as was shown by Head and Thompson (1906), Petrén (1910), and others.

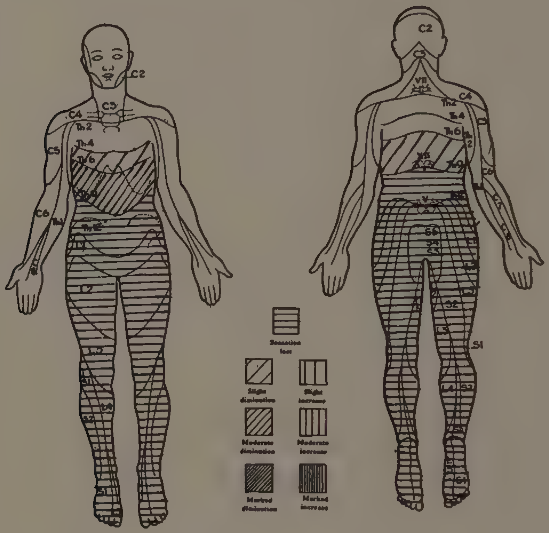
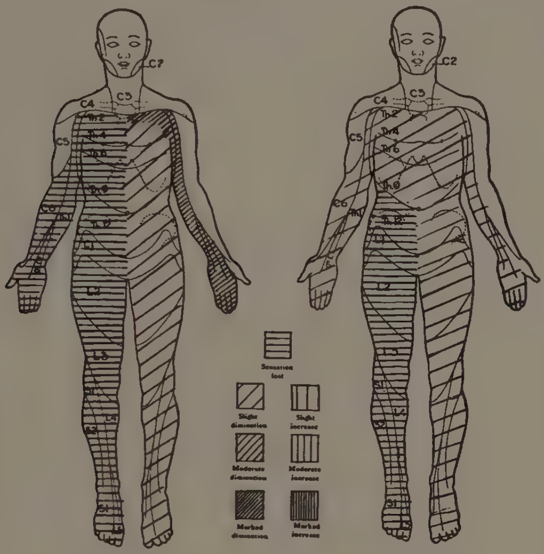


FIG. 197. Cutaneous sensory disturbances in a patient with a tumor on the posterior surface of the cord at the third thoracic segment. The sensory disturbances on the front of the chest were very slight over the fifth and sixth thoracic dermatomes and no disturbances of sensation could be found on the posterior surface of the body over these dermatomes.

There is very often, however, unless there is a loss of all sensation below the segmental level, a striking difference between the sensory disturbances that occur in extramedullary tumors and those that occur in intramedullary tumors.

In most extramedullary growths, and especially in those which lie on the dorsal and lateral aspects of the spinal cord, the maximum of sensory disturbance is in the most peripheral dermatomes, and the minimum of disturbance is nearest the level of the lesion; while in intramedullary growths the maximum of sensory disturbance is often at and below the segmental level. A few of the sensation charts are reproduced in Figures 194, 196, 197, 198, 199.

This difference between the sensory disturbances observed in many intramedullary and extramedullary growths can be explained only upon the basis that the fibers in the pathways for cutaneous sensations are grouped according to limbs and parts of limbs (Fig. 200). The clinical evidence supports the view that in the spinothalamic tracts the most excentric and most posterior fibers are those that are distributed to the sacral and lower lumbar dermatomes, that in front and somewhat internal lie the upper lumbar and lower thoracic fibers, still further forward the upper thoracic, and so on (Fig. 5). A study of many of the cases in the literature gives support to this view.



Pain Temperature

FIG. 198. Cutaneous sensory disturbances showing dissociation between pain and temperature in a patient with a tumor in front of the left side of the cord at the sixth cervical segment.

The variations in the amount and direction of the pressure exerted upon the spinal cord by tumors are so manifold that it is surprising that there are so many cases in which the sensory examination reveals what one could call the typical sensory loss of an extramedullary expanding lesion.

The occasional absence of cutaneous sensory diminution over several or a number of dermatomes below the segmental level can also be explained on the basis of a pressure from behind which has not yet been transmitted to the most anterior part of the spinothalamic tracts.

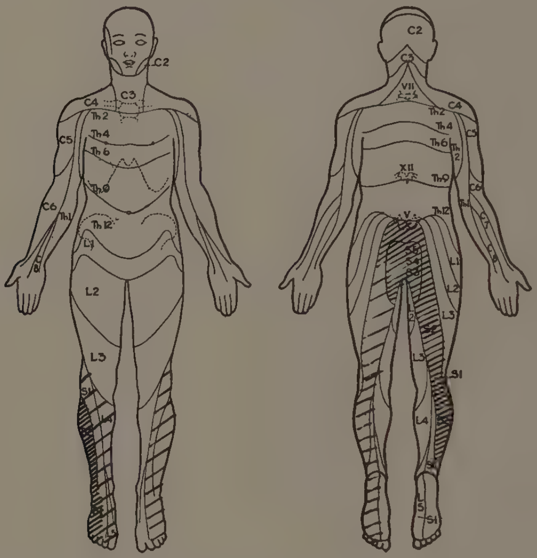


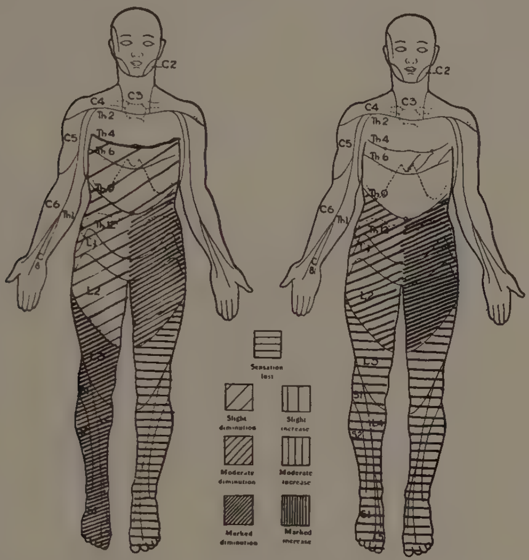
FIG. 199. The cutaneous sensory disturbances in a patient with an ependymoma of the conus and cauda equina.

It is not rare to find, as Heilbronner (1908) observed, an area of normal sensation between a band of root hyperalgesia or root anesthesia, and the main area of diminution or loss of sensation. This has led to errors of localization, especially in those patients in whom the root signs were ill defined or wanting.

In fact, in a large number of patients with the signs of a moderate compression of the cord, there is, above the level of well-marked sensory loss, an area in which the cutaneous sensory disturbances are very slight, so that they merge gradually with the area of normal sensation. This area of slightly disturbed sensation may extend over a number of dermatomes and is easily missed.

Babinski and Jumentié claimed that within this area the sense of localization is decidedly diminished, as compared to that in the segmental skin areas above the level of the lesion.

In pressure on the cord from in front, the fibers which run in the most posterior portions of the tracts may not be involved when the examination is made, and therefore lower skin areas may remain undisturbed. Cases of this kind have been reported by Barré and Jarkowski (1910), Karplus (1914), Collins and Marks (1915), Foerster (1921), and others. Foerster, in his discussion of a case of a tumor at the eleventh thoracic level, states that in this patient there was, for a time, an incongruity between the spastic paralysis of the lower extremities and the loss of the lower and middle abdominal reflexes, on the one part, and the sensory disturbance that extended only up to the groin, on the other. He states that at the level of the tumor, the anterolateral tracts were evidently not yet involved in their entire thickness, "so that the long fibers of the eleventh and twelfth thoracic areas, which lie most internally, were not yet affected by the pressure, while the fibers which corresponded to the



Touch Pain and Temperature

FIG. 200. Cutaneous sensory disturbances in a patient with a spinal cord tumor giving evidence of lamellation in the sensory pathways. Sensation is most disturbed in the most caudal dermatomes and least disturbed in the most cephalic ones.

lumbar and sacral segmental zones which lie more externally had already succumbed to the pressure."

Barré and Jarkowski (1910), Karplus (1914), Head (1920), Babinski, Thomas, as well as Foerster, remark that the sacral segments may often be spared in compression of the spinal cord from without. Head, who is also quoted by Karplus, says that there must be a lamellar arrangement of fibers in the spinothalamic tracts by which the freedom of disturbance in the sacral dermatomes in some cases can be explained.

Serko (1914), in an excellent paper on the subject of cord tumors, states that in extramedullary compression, sensation becomes diminished in an ascending direction, and returns, after the removal of the tumor, in a descending direction.* Serko believes that in the spinothalamic tracts, the fibers for the lower parts of the body lie most external, an arrangement directly opposite to that of the fibers in the posterior white columns. In intramedullary tumors, on the other hand, sensation is diminished in a descending direction. He makes an additional interesting statement which I have been able to substantiate: that if a growth happens to lie in the median line on the front of the spinal cord, there may be a descending sensory disturbance and the sacral dermatomes may be spared.

Whether this sparing of the sacral areas in such a medianly placed anterior tumor is due to involvement first of the median fibers in the anterolateral columns (as Serko believes), or whether it is the result of a pressure that has not been as yet transmitted to the most posterior (and external) fibers in the pathways, is an open question. The fact that it may occur is what interests us, because it is one of the explanations why a ventrally placed tumor so often presents the clinical picture of a growth within the substance of the cord.

Unless the patient comes under observation when all cutaneous sensation below the level of the lesion has already been abolished, there are usually some differences between the sensory changes on the two sides of the body. In a large number of the patients with extramedullary growths, there was more sensory loss on one side of the body than on the other, usually on the side opposite to that on which the tumor lay. This greater disturbance of cutaneous sensibility usually involved all three sensations—touch, pain, and temperature.

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* He adds in a footnote that motor disturbances often progress and regress in the same direction.

Tactile sensation is rarely more affected than pain and temperature sensibility. Unless all cutaneous sensation has been lost, the tactile disturbance is either less marked or is affected equally with the other superficial sensibilities. There are exceptions to this rule, however.

Pain and temperature sensibility are sometimes equally diminished; at other times temperature sensibility is more disturbed than pain, and in these patients pain sensibility may be only moderately disturbed, while the power to recognize and distinguish between extremes of temperature is lost entirely.

There is often a marked dissociation between the ability to recognize hot and cold, and in many cases the patient is unable to recognize heat at a time when he is still able to recognize extreme cold. If this is the case, however, the patient is easily confused and will make many errors even in the recognition of cold. In some instances heat is not recognized at all, but cold, though not recognized as such, is distinctly painful. The patients usually state that the sensation is a "stinging" or "burning" pain, a real pain and not a disagreeable sensation only.

It is a very interesting fact that this perversion of the sensation of cold in which cold is not recognized as such, but is felt as pain, may be found over areas in which pain as tested with a sharp needle and even by deep pressure, has been entirely abolished.

Finally, mention must be made of the fact that in those cases in which the cord is pushed to the opposite side of the spinal canal and the spinothalamic tracts of that side are compressed against the bony wall of the spinal canal, the most advanced sensory changes may occur on the same side as that of the tumor ("reversed" Brown-Séquard syndrome). (See Fig. 196.)

In tumors that involve the conus and the roots of the cauda equina, the sensory disturbances are usually bilateral and asymmetrical, and generally affect all types of sensation to an equal degree.

In extradural tumors, the sensory changes may be ill defined for a long period, due to the fact, to which attention was called by Gehuchten and Lambotte (1913), that the pressure of an extradural growth is apt to be diffuse. This is due to the fact that the spinal cord is for a long time protected by a fluid water pad underneath the arachnoid. Definite sensory disturbances (and also motor loss) often appear rather acutely, when the tumor suddenly overcomes the fluid and impinges upon the cord. The same condition often follows the withdrawal of cerebrospinal fluid by lumbar puncture. In general, the diminution or loss in sensation is very similar to that which occurs in extramedullary growths, but the "reversed" Brown-Séquard syndrome, (more sensory disturbances on the same side and more motor disturbances on the opposite side to that of the tumor) is relatively frequent.

In intramedullary tumors, the sensory changes may be very similar to those in extramedullary growths, and, as already mentioned, typical dissociation of tactile and of pain and temperature sensations is not often found unless the patients are seen early in the course of their disease. Relatively, however, dissociation is more frequent in intramedullary than in extramedullary growths. The sensory changes are not rarely more marked on one side, and in some patients the tactile sensation is involved only on one side, while the power to recognize painful and temperature stimuli is more affected on the opposite side. There is the same tendency for temperature to be more affected than pain sensibility.

There is no more valuable aid for the determination of the relation of a spinal tumor to the sides of the cord than the investigation of the changes in the so-called deep sensibilities, and more especially in *vibratory* and *articular* sensation. A loss, or greater diminution of vibratory and articular sense in one extremity or one side of the body than in the other, is certain evidence, in the large majority of instances, that the growth is on that side of the cord. Disturbance in these two deep sensibilities usually go together, but in some patients vibratory sensation is undisturbed while articular sense is affected, or vice versa.

As was to be expected, in a few patients with the motor and sensory picture of the "reversed" Brown-Séquard syndrome, the disturbance of deep sensibility was on the side contralateral to the tumor but homolateral with the greatest motor loss.

Disturbances in vibratory and articular sense will always occur when the pressure has affected the entire thickness of the cord, no matter whether the growth is ventrally or dorsally placed and no matter whether the growth is extradural, intradural, or intramedullary. These deep sensibilities are obviously always lost when there are the signs of a complete transverse lesion of the cord.

Near the upper level of the lesion, the vibration of a tuning fork is transmitted upward from an area in which this variety of deep sensibility is lost to an area in which it is preserved, and the patient usually states that he feels a vibration before the actual upper level of vibratory loss has been reached. The results of the examination of this type of sensibility, as far as the exact level of the lesion is concerned, are of little practical value. The closest approximation of the upper level of vibratory disturbance to the upper level of cutaneous disturbance is found when the tuning fork is successively placed on the tips of the spinous processes of the vertebrae, and when a tuning fork with a high frequency of vibration (C 256) is used.

Diminution or loss of vibratory and articular sense is regularly met with in extramedullary tumors on the dorsal or dorsolateral aspect of the cord, and sometimes occurs in ventral and ventrolateral growths. In several patients with tumors in front of the cord, the diminution or loss of vibratory and articular sense led us to believe that the tumor lay on the posterior aspect of the cord. At operation, however, the tumor was found on the anterior aspect, the cord having been pushed backward by the growth so that the posterior white columns were pressed against the dura and the posterior wall of the vertebral canal and the impulses through the long fibers in the posterior columns interfered with.

THE COMBINATION OF MOTOR AND SENSORY DISTURBANCES: THE BROWN-SÉQUARD SYNDROME AND ITS VARIATIONS

The typical combination of motor and sensory disturbances due to interference with the functions of one lateral half of the cord, as described many years ago by Brown-Séquard and ever since known by his name, is rarely seen in a complete form in spinal compression by a new growth.

In what follows, I shall often speak of a Brown-Séquard type of motor and sensory disturbance, because it best describes a combination of signs in which the most marked motor disturbances are on the same side as the tumor, and the most marked cutaneous sensory disturbances on the opposite side. This is done, in spite of the fact that in a true Brown-Séquard syndrome, there are motor symptoms on only one side, and pain and temperature disturbances on the opposite side as an evidence of an affection of only one lateral half of the cord. In spinal tumors, the lesion is seldom anatomically limited to one-half of the cord, but very often one side of the cord is much more affected than the other side.

In many of the extramedullary and extradural tumors, there was some indication of a Brown-Séquard type of motor and sensory disturbance, although often it was poorly marked. There was generally more motor disturbance on the same side as that of the tumor, and more sensory changes on the opposite side. In intramedullary growths, on the contrary, the indication of a Brown-Séquard type of motor and sensory involvement was seldom as distinct as in extramedullary growths.

Karger (1916), in reporting a case of intramedullary spinal tumor with a typical Brown-Séquard picture, declares that this is rare in growths within the substance of the cord; and he quotes Malaisé (1904), who declares that up to the year 1904, there were only two other cases known, those of Henneberg and of L. R. Müller (1897).

In not a few patients, cutaneous sensation was equally diminished on the two sides of the body, while motor power was more affected on the same side as that of the tumor.

In a number of patients in my series, we observed what I have called a "reversed" Brown-Séquard picture, in which the motor disturbances were most marked on the side opposite to that of the tumor, and the sensory changes frequently most marked on the same side. The explanation for this anomalous condition is that in some extradural and intradural growths adherent to the dura, the cord soon occupies a position on the opposite side of the vertebral canal, so that the cord on that side is pressed against the dura and bony wall of the canal with symptoms primarily due to involvement of the motor and sensory pathways on that side of the cord. When bilateral signs become marked, the condition may then be one of a "bilateral Brown-Séquard symptom complex."

FREQUENCY AND LOCATION OF TENDERNESS OF THE SPINOUS PROCESSES OF THE VERTEBRAE IN CORD TUMORS

Tenderness of spinous processes upon percussion or upon pressure is very frequent, and occurs not only in extradural and extramedullary (intradural) but also in intramedullary growths.

The spines that are sensitive are usually not at the vertebral level of the tumor, but one, two or three spines below that level. For example, with a tumor at the sixth thoracic segment, the spinous process of the sixth thoracic vertebra was sensitive to pressure; with a tumor at the twelfth thoracic segment, the spine of the twelfth thoracic vertebra, and perhaps that of the first lumbar vertebra, was sensitive. In other words, in extramedullary tumors, if pressure upon any spinous process is painful, it is the spine of the vertebra which receives its nerve supply from the posterior spinal root of the affected segment of the cord, and the tenderness is probably due to irritation of that sensory root by the manipulation of the spinous process. It is, of course, the posterior root which lies in close proximity to, or is actually involved by, the tumor.

In tumors of the cervical cord, the spinous process tenderness is apt to be more diffuse, so that a number of the cervical spines are tender. In extradural growths, especially in those that arise from the vertebrae themselves, the tender spines usually correspond to the vertebrae affected, and there is a true bone tenderness from the osseous lesion. This difference of location of the spinous processes that are sensitive, is of value for the differentiation between cord tumors and extradural malignant bone disease.

Some writers have claimed that tenderness on pressure over the laminae and transverse processes to one side of the spine occurs especially in bone disease, but I have not been able to substantiate this claim.

CHAPTER X

TUMORS OF THE SPINAL CORD, NERVE ROOTS, AND MEMBRANES

IV. DIAGNOSIS

CLINICAL FEATURES AT DIFFERENT LEVELS

FROM THE STANDPOINT OF THE PHYSIOLOGY OF THE SPINAL CORD, THE DISTURBANCES PRODUCED BY PARTIAL OR COMPLETE LESIONS at various levels, are described in the chapter on fractures and dislocations of the spine, to which the reader is referred.

At different levels of the cord, the symptoms and signs produced by tumors vary considerably with the size, especially with the longitudinal extent, of the growth. A small tumor may make pressure upon only one nerve root and upon one or two cord segments, while a larger one may affect several nerve roots, and three, four, perhaps more, cord segments.

On the other hand, although a growth may have considerable length, its thickness may vary in different parts. The upper or the lower end of the growth may have the greatest thickness, or the transverse diameter of the middle of the growth may exceed that of the upper and lower poles. As a result, different parts of the tumor may exert a different amount of pressure, or only one part of the growth may actually compress the cord.

There is, therefore, a marked variation in the combination of symptoms, and this depends upon the number of segments affected by the growth. When a tumor is described as lying against or extending over a certain number of segments of the cord, it does not mean that all of this part of the cord is actually involved by the growth, or that of necessity the signs of cord disturbance extend as high as the tumor itself. This accounts for the discrepancy between the level signs of some growths and the exact location in which they are found. Although spinal tumors occur at every level of the cord, and although they vary considerably in size and shape, still there are certain clinical pictures most often encountered. These are described in the following pages.

1. Tumors at the First to the Third Cervical Segments

These can be grouped together. They usually begin with pain in the neck and occipital region, although, if the tumor has begun at the third or the fourth cervical segment and has increased in size in an upward direction, the first pain may be felt at the root of the neck, or on the top of the shoulder corresponding to the fourth cervical area. The pain is usually severe and made worse by movement, and as a result, the patient holds his head more or less stiffly. In the only high intramedullary growth in my series, the early symptoms were a numbness in one cheek and attacks of "shooting" pain in the side of the face.

Soon thereafter, there is an increasing weakness of the upper limb on the side on which the pain was most marked. The weakness may be first noticed in the thumb and index finger, or it may involve, from the beginning, more or less of the entire extremity. It is soon followed by subjective and objective sensory disturbances in the same limb.

Sometimes the patient complains of a feeling of coldness in one or both upper extremities, but much more often there is a feeling of intense coldness in one or both lower limbs. A sensation of tingling in the contralateral lower extremity was complained of by a number of my patients with high cervical cord tumors, and sometimes there was a sensation of constriction around the neck.

Attacks of difficulty in breathing and of a feeling of suffocation are complained of sooner or later. This difficulty in breathing may occur only late in the disease.

Loss of power and stiffness or wasting of one upper extremity are usually noted by the patient for a considerable period before any other part of the body becomes affected.

Increasing weakness and stiffness of the lower limb of the same side next makes its appearance, and at this stage, the patient may have a true "spinal" hemiplegia. The contralateral lower limb and then the contralateral upper limb becomes involved in the loss of power, until finally the patient has a tetraplegia.

Cranial nerve symptoms are surprisingly infrequent in these high cervical growths and signs referable to cerebellar disturbance are unusual. Nystagmus was present in 4 patients of my series. The fundi oculi were normal in every instance excepting that in 1 patient the retinal veins were somewhat congested, and in another, there was definite papilledema. Papilledema caused by cervical cord tumors has been reported by McAlpine (1935), and by others.

The pupils reacted well to light and accommodation in every patient, but in 1 case there was an inequality of the pupils.

Hyperesthesia of the cornea was noted in 1 patient and a diminution of sensation over the distribution of one trigeminus in another. Subjective fifth nerve disturbances occurred in both patients.

Weakness of the facial muscles on one side is not very frequent; it was found in only 2 patients.

There was no deviation of the tongue in any of my patients, and I have been able to find in the literature only one case of upper cervical cord tumor with hypoglossal nerve symptoms (Gardner, 1938).

The phrenic nerves (from the fourth cervical segment) may not be involved in high cervical lesions, especially if the lesion is not advanced. In 2 of my patients, fluoroscopy showed a normally acting diaphragm on both sides of the body. Oppenheim (1913) called especial attention to this, Auerbach and Brodnitz, Antoni, and others have reported similar cases.

Atrophy or spasm of the sternomastoid and trapezius muscles is not rare, and fibrillary twitchings of these muscles are sometimes observed.

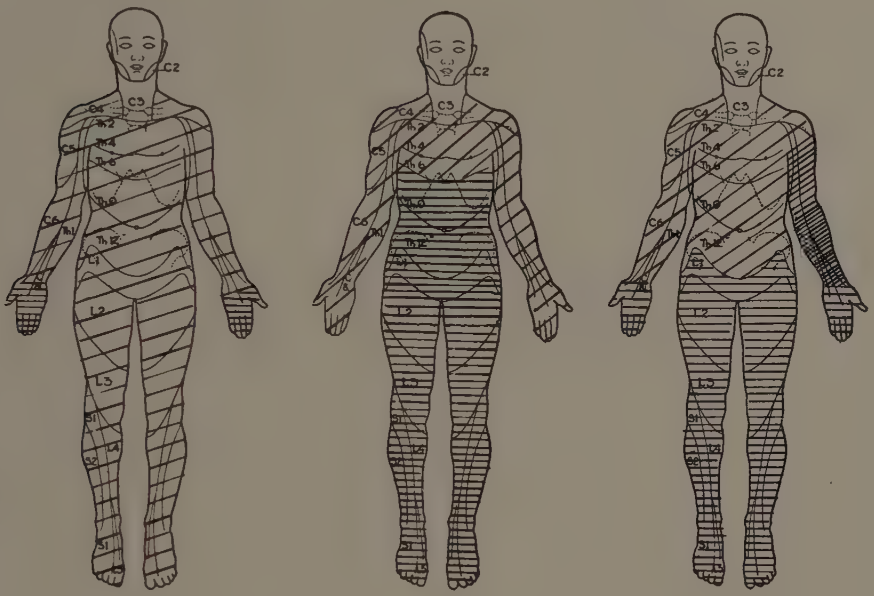
Atrophy of the muscles of the shoulder girdle and of all the muscles of one or both upper extremities is very frequent. Thus in one patient, there was wasting of the pectoral, deltoid, supraspinatus and infraspinatus, biceps and triceps muscles; in another case there was "considerable atrophy of both upper limbs"; in another, the atrophy involved the trapezius and sternomastoid, the muscles of one shoulder girdle and of the entire upper extremity; and in still another, the entire upper limbs, with especially well-marked wasting in the intrinsic musculature of the hands. Such extensive muscle atrophies in high cervical cord tumors have been reported by Oppenheim (1913), Söderbergh (1914), Henneberg (1921), and others. It is noteworthy that this wasting involved muscles whose anterior gray horn cells lie in segments below the part of the cord that is compressed, and the clinical picture may be very similar to that of amyotrophic lateral sclerosis.

There is nothing especially characteristic about the reflexes in the upper and lower extremities. The tendon reflexes in one or both upper limbs may be diminished or absent if there is marked muscle atrophy, or they may be exaggerated if there is marked hypertonicity.

The sensory disturbances in these cases of high cervical tumor vary within wide limits. The diminution or loss in sensation may involve the back of the head, the neck, the top of the shoulders and the entire body. More often, however, there is hyposensitiveness in some part or all of the second, third, and often the fourth cervical dermatomic areas. In high cervical extramedullary tumors, there is, not so rarely, dissociation between cutaneous sensibilities. Very frequently, tactile sensation is only slightly involved while pain and temperature sensibilities are greatly diminished.

2. TUMORS AT THE FOURTH TO THE SIXTH CERVICAL SEGMENTS

The symptoms of extramedullary tumors at the fourth to sixth cervical segments (Figs. 201, 202) most often begin with pain in one shoulder or in the entire upper extremity. At times, however, the first symptoms are referred to the thumb and index finger of one hand or to the root of the neck. Extramedullary tumors in this location, no matter what their relation to the cord, usually begin with root symptoms, which often persist throughout the course of the disease. In intramedullary growths at these levels early pain is also frequent.



Tactile Pain Temperature

FIG. 201. Cutaneous sensory disturbances in a patient with an extramedullary tumor at the level of the fourth to the fifth cervical segments.

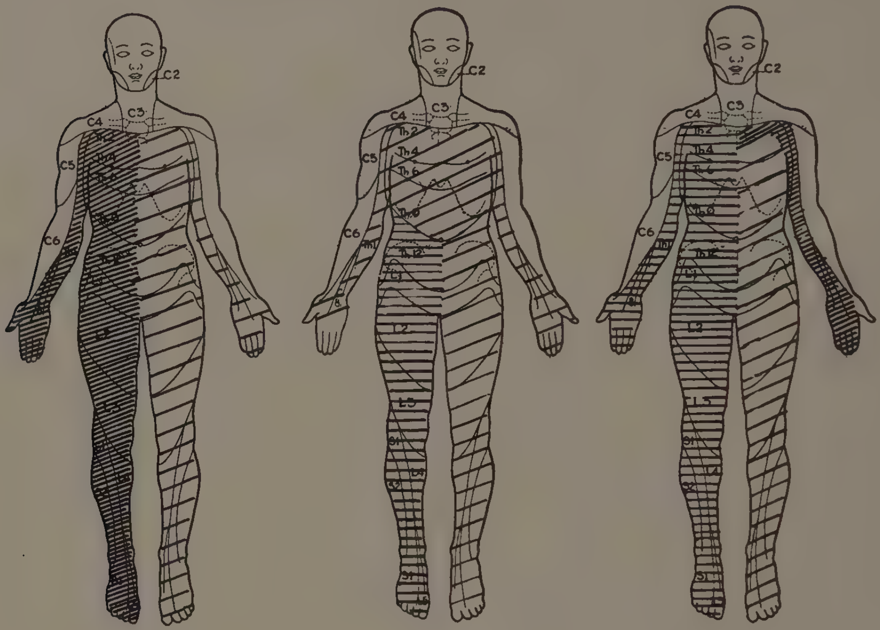
The pain is followed sooner or later by a loss of power in one upper extremity and later in the homolateral lower limb.

In extramedullary growths at the sixth cervical segment, there is a period during which motor weakness involves only the upper and lower limb of one side, and it is in these patients that one is justified in speaking of a stage of "spinal hemiplegia." After a longer or shorter period, the limbs on the opposite side become weak, and this loss of power most often first involves the contralateral lower limb.

In intramedullary tumors, however, both upper limbs (and both lower) may be involved at the same time, although unilateral weakness is the more frequent.

The loss of power in the upper extremities usually involves the muscles of the shoulder girdle and all the muscles of the upper extremity. Depending upon the stage in which the patient is examined, the weakness may involve one or both limbs, though the homolateral one is most markedly affected.

In all our patients with growths at this level, there was atrophy of



Touch Pain Temperature

FIG. 202. Cutaneous sensory disturbances in a patient with a tumor on the ventral and left lateral aspect of the cord at the sixth cervical segment.

the intrinsic muscles of the hand, although the segments actually compressed by the growth were often above the spinal centers for these muscles. If the fourth and fifth cervical segments bore the brunt of the pressure, the wasting was usually well marked in the muscles of the shoulder girdle—deltoid, biceps, supraspinatus and infraspinatus, subscapularis and clavicular part of the pectoralis major; while if the pressure was exerted especially upon the fifth and sixth segments, the entire pectoralis or only its costal portion was involved and the atrophy of the deltoid, biceps, and brachialis anticus was especially prominent.

The flexor (and extensor) power at the elbow is often diminished or lost and abduction and adduction at the shoulder joint may be weak. Extension of the fingers may be impossible, or both flexion and extension of the first three or all of the fingers are lost. There may be typical *main-en-griffe*.

There is considerable variation in the tendon reflexes. In the typical cases, the biceps reflex on one or both sides is diminished or lost, while the triceps reflex is exaggerated. In some patients neither of these reflexes can be elicited.* The radius reflex (contraction of the supinator longus and perhaps of the biceps and flexion and slight supination of the forearm on percussion over the styloid process of the radius) is often lost with the biceps jerk, but it is curious that the radius reflex may be hyperactive at the same time that the biceps jerks cannot be elicited. I have several times observed, when the styloid process of the radius is tapped with the percussion hammer, the flexion movement of the fingers first described by Babinski. The ulnar reflex (pronation of the forearm on percussion over the lower end of the ulna) is usually preserved unless the triceps jerk cannot be elicited. In such a case the ulnar reflex is also lost.

In several patients when the fingers were extended and the hand was dorsally flexed there was a well-marked clonus at the wrist. This has also been observed by Oppenheim.

There is nothing especially characteristic for this level in the changes of the cutaneous and tendon reflexes in the trunk and lower extremities.

The sensibility disturbances observed in tumors that extend a varying distance downward from the fourth cervical segment are subject to great variation. There may be no changes in the fourth cervical dermatome, although the tumor extends over it. In other cases there is hyperesthesia above and below the clavicle or on the top or outer aspect of the shoulder, on one or both sides, sometimes with hyperesthesia over the outer aspect of one or both arms and forearms.

If the uppermost level of the tumor is at the sixth cervical segment, the highest sensory disturbances may consist of hypersensitiveness, or diminution or loss of cutaneous sensibility over the outer aspect of the lower end of the arm and all of the forearm.

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* This means, of course, that either the pressure of the tumor has been transmitted to the seventh (and eighth) cord segments, or that there is an edema or a vascular change in that part of the cord.

3. Tumors at the Seventh Cervical, the Eighth Cervical and the First Thoracic Segments

Tumors at the seventh and eighth cervical segments often extend downward over the first thoracic or upward to the sixth cervical segment. Sometimes they begin with pain which extends down the inner side of the arm to the fingers; at other times, if there is any pain at all, it is first felt in the fourth and fifth fingers of one hand. Intramedullary growths at this level may begin with unilateral or bilateral pain or paresthesias over the same areas; or the first disturbance noted is atrophy of the intrinsic muscles of one or both hands.

The order in which loss of power in the extremities occurs most often follows the usual formula: homolateral upper limb, then lower limb, followed by weakening of the contralateral lower, and finally the contralateral upper limb. But the contralateral limbs are affected much more quickly than in upper and middle cervical cord compression, and in intramedullary growths the corresponding limbs of the two sides may be involved at the same time.

The small muscles of the hand and the triceps and sometimes the extensor longus digitorum and the extensor longus pollicis, become atrophied. Oculopupillary symptoms (*vide infra*) are frequent. The weakness in the upper extremity involves mainly the extensor power at the elbow and the movements of the wrist and the fingers. *Main-en-griffe* is not unusual when the patient is seen in an advanced stage, and clonus of the extensor muscles of the forearm may follow forced flexion at the wrist.

The biceps reflex is preserved while the triceps is weak or lost, or there may be an inversion of the triceps reflex. The radius and ulnar reflexes may be diminished or lost or the radius reflex may persist. There is considerable probability that the cord centers for the radius reflex are not always located in the seventh and eighth cervical segments, but sometimes in the sixth, and perhaps also in the fifth, cervical segment.

If the pressure is at the eighth cervical and first thoracic segments, and involves the roots, the Déjérine-Klumpke type of paralysis is often seen, in which especially the flexor muscles in the forearm and the intrinsic musculature of the hand are affected.

If the pressure of the tumor is exerted upon the cord no higher than the first thoracic segment, the motor disturbances in the upper limb are usually not well marked. The triceps is seldom involved and extension of the forearm is not interfered with. There may be, however, some weakness of the interossei and lumbricales, especially those of the fourth and fifth fingers, which may be in the typical flexed position so characteristic of lesions of the ulnar nerve.

Disturbances in the oculopupillary reflexes occur when the center for this reflex, which is located in the eighth cervical and first thoracic segments of the cord, is involved. These disturbances consist of diminution in the size of one pupil and of the palpebral fissure (spinal miosis, anisocoria, and enophthalmos) and when combined with vasomotor disturbances (redness or pallor of the skin with hyperidrosis or anidrosis) form the so-called Horner's syndrome. These disturbances occur because the fibers from the ciliospinal center in the eighth cervical and first thoracic segments of the cord pass to the inferior cervical ganglion of the sympathetic mainly through the first thoracic root. Horner's syndrome is often incomplete, and there may be a dissociated oculosympathetic disturbance, with ptosis and enophthalmos and vasomotor and secretory changes, but without anisocoria.

Hyperesthesia over the distribution of the seventh and eighth cervical roots is rarely seen, excepting in intramedullary tumors, but hypersensitiveness over the first thoracic dermatome does occur. If the growth affects the cord as high as the seventh and eighth cervical segments, the sensory disturbances involve the inner aspects of both upper extremities from the third, fourth and fifth fingers to the inner aspect of the arm in the axilla. If the first thoracic segment is the highest level, diminution or loss of cutaneous sensibility on the limb usually does not extend below the middle of the forearm.

There is nothing especially characteristic in the disturbances of cutaneous sensibility on the trunk and lower extremities. In pressure between the seventh cervical and first thoracic segments, disturbance of vibratory sensibility—if there is any change at all—can rarely be found on the anterior surface of the body higher than the free costal border.

There is one aspect of the cutaneous sensory disturbances in tumors of the middle and lower cervical segments that is worthy of especial mention: tactile sensibility may be relatively little involved as compared with pain and temperature sensibility. This we have observed not so rarely in patients with cervical cord tumors, especially if the growth lay on the ventral or ventrolateral aspect of the cord.

In a recent case of tumor at the fourth cervical segment, no disturbance of tactile sensibility could be discovered, and the changes in the ability to recognize a pin point and the hot and cold test tube were not well marked. Had it not been that in other respects the history and progress were typical, the diagnosis of extramedullary cord tumor could not have been made. This absence of distinct tactile disturbance is of interest, and should not mislead one if other signs point to progressive compression at a definite level.

4. Tumors at the Second to the Eighth Thoracic Segments

These often begin with pain, which has the character of an intercostal neuralgia, in the back or chest on one or the other side. The pain may begin on one side and spread to the other, or may begin on both sides and finally become localized on the right or on the left. In upper thoracic tumors, the patients often state that the pain began under one shoulder blade. When the tumor lies between the fifth and eighth thoracic segments, the first pain may be felt underneath the costal border, and if the pain is on the right side and occurs in attacks, the symptoms may simulate cholelithiasis or a gastric disturbance. In a relatively large number of patients, subjective sensory disturbances may first occur in one or both lower limbs. In intramedullary growths at the second to eighth thoracic segments, the onset is either without pain or other subjective sensory symptom, or the paresthesias occur first in the lower extremities.

Motor and sensory changes then follow in the lower limbs, and finally disturbances in the functions of the bladder and rectum.

In the higher thoracic growths, a girdle sensation around the middle or lower part of the chest is frequent; while in the growths of the sixth to eighth thoracic segments, the girdle sensation, if it occurs at all, is felt around some part of the abdomen.

In the majority of cases, the abdominal and the cremasteric reflexes (in the male) are lost relatively early, but the upper abdominals may remain weak, while the lower are lost; or, vice versa, the lower may persist while the upper disappear. In the early stages, the tone of the abdominal muscles may be much increased; in the more advanced stages, the power of the muscles is usually distinctly diminished.

The upper level of the sensory disturbances depends upon the location of the growth. Upon careful examination, a band of root hyperesthesia or root anesthesia is often found, which extends all around the body at the upper level of the sensory disturbances; and in advanced cases root hyperesthesia with loss of all cutaneous sensibility below the affected level is not rare.

In some patients with a tumor at the fifth or sixth thoracic segment, the sensory disturbances above the lumbar dermatomes are extremely slight and easily missed. If this should be the case in a stout individual, in whom no abdominal reflexes can be elicited on account of the adiposity, there would be danger that the disease would be sought for at the upper lumbar levels, when it was really in the midthoracic region, or that it would be considered to be intramedullary on account of the relatively slight diminution of tactile sensibility in the lumbar and sacral dermatomes. An important finding, however, and one that makes intramedullary disease in the lumbosacral cord improbable, is a disturbance of vibratory sensibility which extends up to the anterior superior spines of the ilia.

Babinski and Jarkowski have made a very valuable contribution to the study of the sensory disturbances that occur in compression of the thoracic cord. I can substantiate every one of their statements from my own experience, and it may be of interest to quote some parts of their paper.

The authors declare that in complete transverse lesions of the thoracic cord, there is often, above the area of complete anesthesia, a zone in which sensation is so much diminished that the patient cannot distinguish between the head and the point of a pin, and cold is felt as if it were a pin prick. This zone is usually 3 to 4 cm. wide. Above this is another zone which is easily missed by the examiner, in which cutaneous sensibility is preserved but is very slightly diminished.

In incomplete lesions, on the other hand, there is the same area of diminished sensation, but the disturbance seems to vary during the examination. "L'anesthésie instable occupe souvent, dans les compressions spinales, la plus grande partie ou même la totalité du territoire interessé."

Thus, there may be an area which extends over a number of segmentary areas, in which the sensory disturbance is very slight.

5. Tumors at the Eighth, Ninth, Tenth, Eleventh, and Twelfth Thoracic Segments

In these patients, pain, if present, is felt on the surface of the abdomen and sometimes in the abdominal cavity. In tumors between the tenth and twelfth cord segments, the pain is often felt around one hip or at the crest of the ilium, and sometimes radiates down one or the other lower extremity. Early pain and stiffness in the lower part of the back are also frequent complaints. Subjective sensory disturbances in the lower extremities are not rare.

The motor disturbances involve the muscles of the abdomen and the lower extremities. Unilateral weakness of the abdominal muscles, very noticeable when the patient attempts to strain, often occurs. The upper abdominal reflexes have their spinal centers in the eighth and ninth, and the lower abdominal reflexes, in the tenth, eleventh, and twelfth (?) thoracic segments. Therefore when a growth is located at the eighth and ninth segments, one or both of the upper abdominal reflexes may be weak or lost altogether, in addition to the lower abdominals and cremasters. If the tenth to twelfth segments are involved, the upper abdominals may be preserved while the lower, on one or both sides, are lost. I believe it probable that the lower abdominal reflex has very little representation in the twelfth thoracic segment. In cases of injury or tumor that do not extend above the twelfth thoracic segment, the lower abdominal reflexes are usually preserved.

In these tumors at the lower thoracic levels the root symptoms may be erroneously interpreted as signs of an abdominal lesion. In these patients a laparotomy for supposed intra-abdominal disease has sometimes been done. For a long period, the motor and sensory disturbances in the lower limbs may be slight and limited to a moderate overactivity of the Achilles reflexes and vague sensory diminution on the dorsal and plantar surfaces of the feet.

Xanthochromia is frequent in compression of the lower thoracic cord, and *eo ipso*, a deep yellow fluid suggested a tumor at or below these levels.

6. Tumors at the Upper Lumbar Segments

Except for the difference of the level, the clinical features of tumors at the upper lumbar segments do not differ essentially from the symptoms produced by growths at higher parts of the cord, unless the growth extends downward and involves some of the roots of the cauda equina. If the patient has pain at the onset of the disease, the pain is generally referred to the back or to some part of the lower limbs. Sometimes the pain is felt in the lower abdomen, in the bladder, or in the rectum. If the tumor compresses the first or first and second lumbar segments, but does not involve the twelfth thoracic segment, the abdominal reflexes are preserved, the cremasteric reflexes disappear, and the knee and ankle jerks are hyperactive. If the tumor affects the second, third, and fourth lumbar segments, the cremasteric reflexes may be preserved, while one or both knee jerks are diminished or lost, and the Achilles reflexes are preserved or exaggerated. The clinical combination of loss of knee jerks and ankle clonus is occasionally met with, but more often, on account of the relatively small extent of the lumbosacral cord, knee and ankle jerks are diminished or lost together.

If the cord at these levels is compressed without involvement of the roots of the cauda equina, one or both lower limbs may be spastic, but more often the paralysis is a flaccid one. There may be a flaccid paralysis of one limb due to involvement of the roots of the cauda equina, and a spastic paralysis with hyperactive knee and ankle jerks, and perhaps ankle clonus of the limb of the other side. The segments of the lower lumbar cord lie so close together that few, if any, sensory fibers can cross to the other side below a tumor in this location. As a result, sensory and motor disturbances may occur on one and the same side. As a fact, however, sensory and motor disturbances are usually bilateral and often asymmetrical.

7. Tumors of the Conus and the Cauda Equina

These have certain clinical features that are characteristic for these levels of the cord. The conus really does not extend above the third sacral segment, but we are accustomed to speak of these growths as "conus tumors" because they begin primarily over the conus or between the roots of the cauda equina, and secondarily may involve the lumbosacral cord.

The first symptom in these patients is usually pain, often excessively severe, in the small of the back, which sooner or later radiates down one or both limbs. In a few patients, the pain occurs first in a part of one or the other extremity, and occasionally it is first felt in the rectum or in the genital organs. The patients are often treated for long periods for "sciatica." After a period which may extend over many years, there is stiffness of the back in the lumbar region and increasing weakness of the lower extremities with loss of power to flex dorsally one or both feet.

Bladder disturbances may occur early, especially if the growth has originated from the filum terminale, but more often they are late symptoms. Impotence is not rare.

In the typical picture, one or both knee and ankle jerks are lost and there is drop-foot on one or both sides. Wasting of the muscles of the lower limbs regularly occurs, and involves most often the anterior tibial and peroneal groups of muscles. Upon electrical examination, a reaction of degeneration is found in the affected muscles, and fibrillary twitchings are frequently observed.

The sensory disturbances may be slight, but they are usually very marked, involve the lower lumbar and the sacral dermatomes, and are asymmetrical in their distribution. The changes involve all types of cutaneous and "deep" sensibility. Contralateral disturbances of pain and temperature sensibility are rarely seen in tumors of the lumbar or sacral segments, because the fibers that conduct those sensibilities do not decussate until they arrive at about the level of the twelfth thoracic segment.

In some patients there are sensory disturbances in only one or two root areas of one side, which are easily missed unless a careful sensory examination is made. Thus, in a recent case of tumor of the first lumbar root, the only sensory disturbance was moderate hypesthesia over the distribution of the affected sensory root.

It is worthy of especial mention that the large soft tumors of the cauda equina, that often fill up the entire lower end of the spinal canal, and that clinically present very slowly progressive symptoms extending over many years, may cause mainly motor disturbances (Fig. 203). It is difficult to understand that such a large growth may exist in the lower end of the spinal canal without any objective sensory disturbances.

In some cases of tumor of the cauda equina, there is neither subjective nor objective evidence of motor involvement, and the sensory disturbances may be so slight that only a suspicion of hyperesthesia over a root area can be discovered. These cases are often extremely puzzling. In a patient recently operated upon, with a small meningioma between the upper roots of the cauda equina, the only subjective complaint was pain in the small of the back which was increased by sneezing and coughing. Objectively, we failed to find any disturbance of motor power, of reflexes, or of sensation. The persistent pain in the back and the presence of xanthochromia led to an operation and the removal of the tumor with complete relief of the symptoms.

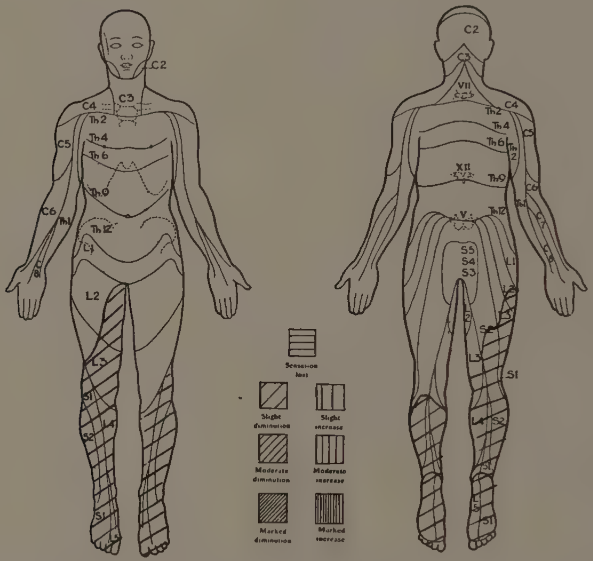

FIG. 203. Very slight cutaneous sensory disturbances in a patient with a very large soft tumor over the conus and roots of the cauda equina.

Tumors that lie between the roots of the cauda equina, especially if the growth is attached to one of the roots, often cause very localized root pains. Root anesthesias are also very common. Tumors adherent to the first lumbar root are especially frequent and usually cause an area of disturbed sensation in one or both groins. The knee jerks may be exaggerated on one or both sides and there may be double ankle clonus, if the tumor has originated from a root near one of the upper lumbar segments. On the other hand, the knee and ankle jerks may be diminished on the side of the growth, and exaggerated on the opposite side.

Vibratory and muscle-tendon sense may be not at all disturbed, and in such a patient, it may be uncertain whether the growth lies in front of the lumbosacral cord, or whether it lies below the end of the conus but has not entirely blocked the affected caudal roots.

Trophic ulcers over the lumbosacral region or the buttocks are very frequent; in fact, the frequent trophic ulcers seen over the sacrum, buttocks, heels and hips in slowly progressive compression of the cord are rare in other parts of the cord, unless the symptoms are very far advanced.

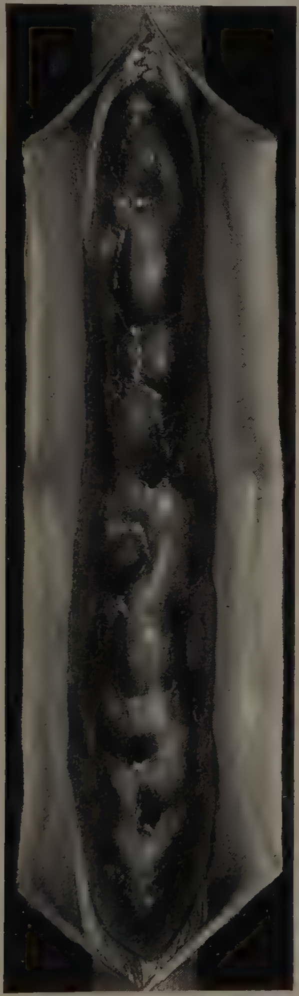

FIG. 204. A very large tumor of the cauda equina exposed at operation. The tumor was an ependymoma.

The infrequency of decubitus in all but the terminal stage of slowly progressive spinal compression is surprising. It is rare to find in a patient with a cervical or thoracic cord tumor a decubitus or other trophic disturbance unless the skin has been injured by coming in contact with excessive heat which was not felt by the patient. This contrasts markedly with what may occur after operative intervention and a certain amount of acute trauma of the cord.

In lumbosacral and cauda equina growths, however, pressure sores and trophic ulcerations of the toes are not at all infrequent.

Special mention must be made of the gross and microscopic characteristics of tumors of the cauda equina. Most of the meningiomas and perineurial fibroblastomas that are found between the caudal roots are not different in size or structure from those found at other levels of the spinal cord. On the other hand, many growths, whose histological classification is often uncertain, are very large and soft. The tumor tissue may be avascular or very vascular. The dura mater over the growth is usually much thinned.

Tumors of the cauda equina are among the largest growths found in the vertebral canal and for this reason I have, for many years, characterized them as "giant" tumors. They often produce a marked enlargement of the spinal canal, which can be recognized on roentgen films and by interpediculate measurements. Many of these large growths are ependymomas which originate either from the filum terminale (Fig. 204) or the conus medullaris, and they can often be completely removed by division of the filum or the conus. In not a few instances, however, the capsule of the growth is either very thin or has ruptured, so that tumor masses surround and are attached to a number of the roots of the cauda equina.

The symptoms produced by the so-called giant tumors are those which are characteristic of all tumors of the cauda equina, but the symptoms of tumors derived from the filum terminale are often characterized by early and persisting disturbances of the functions of the bladder.

DIAGNOSIS

The diagnosis "spinal cord compression by tumor" can often be made without difficulty, especially if the disease has run a typical course. If the clinical investigation has been comprehensive, the errors that have sometimes been made because of a lack of thorough physical examination should be avoided. In every patient with spinal symptoms, the organs in which a primary disease is relatively frequent should come under special scrutiny. When there is a question of multiple growths, the nerve trunks of the extremities should be palpated in the search for multiple neurofibromata.

Careful x-ray examination should always be made, for a hitherto unsuspected bone disease may be discovered; but level symptoms can rarely be caused by arthritic changes in the vertebrae. The measurement of the interpediculate spaces (page 71) should never be omitted.

Equally important are the examinations that should be made in connection with the withdrawal of spinal fluid. A complete examination of the patient should be made and recorded both before the lumbar puncture and after it. Indefinite level signs may become distinct, or existing signs become much aggravated, after the withdrawal of fluid.

Typical cases of cord tumor—those beginning with root pains followed by a slow advance of symptoms of cord compression—form a clinical picture so characteristic that the diagnosis "spinal cord tumor" can be made without difficulty.

In the first stage, when the only symptoms are those referable to one or more spinal roots, the diagnosis may be suspected, but can rarely be made with certainty.

In such a case, however, careful search for slight sensory or motor disturbances below the level of the lesion should be made. If there are more active tendon reflexes on the same side as the root symptoms —no matter how slight—the diagnosis of a growth within the spinal canal is probable.

When the growth involves only a nerve root, the clinical picture is often that of a neuralgia, which involves one or the other limb, or some part of the head, neck, chest, or abdomen. The patients often believe that they are suffering from "rheumatism" or "neuritis." As this neuralgic stage may last for a number of years before there are any signs of interference with the functions of the spinal cord, the condition has often been mistaken for a local affection of a thoracic, abdominal, or pelvic organ, and in many instances useless operations have been performed.

In the *absence of signs of other disease*, a persistent neuralgia must always be looked upon with the suspicion that the pain may be of spinal root origin. This means that the patient may have to be observed for a long period before cord symptoms appear and clarify the clinical picture.

If coughing, sneezing, or other forced expiratory movements aggravate the neuralgic symptoms, the possibility of a spinal root lesion is brought measurably nearer.

Cutaneous hyperesthesia with a distinctly radicular type of distribution points to a spinal root origin of the symptoms, and to this may be added muscular atrophies of the radicular type.

There is often considerable variation in the onset and the progress of cord symptoms, but if the mechanical effects of slowly increasing local cord compression are thoroughly understood, many clinical pictures, which at first seem bizarre and unusual, will be found to be easily explainable.

Although the beginning of spinal symptoms is usually a gradual one, the onset may be sudden, for a growth may slip out from under a nerve root or the dentate ligament, and suddenly make pressure upon the cord.

Any patient with a slowly progressive spinal disease with level sensory signs must be considered a tumor suspect, especially if the history of the onset and progress of the disease and the physical signs of disturbed function cannot be satisfactorily explained by a different disease.

Although the entire clinical picture is often so characteristic that no other diagnosis than that of spinal cord tumor is reasonable, the following conditions may have to be excluded: gumma or syphilitic meningitis, Pott's disease, primary or secondary malignant disease of the vertebrae or of the soft tissues around the bony spine, benign tumors of the vertebrae or intervertebral discs, spondylitis, pachymeningitis, circumscribed serous meningitis, adhesive arachnoiditis, multiple sclerosis, spinal gliosis or syringomyelia, and varices of the spinal blood vessels. In tumors of the lumbosacral cord and roots of the cauda equina, neuritis of the caudal roots must be excluded. Most of these conditions are discussed in detail in other chapters of this book.

Syphilitic disease of the spinal cord and membranes must be excluded in every case of progressive spinal disease, by the examination of the patient's blood and spinal fluid for the Wassermann reaction (Chapter XII). Even in a patient with syphilis and a positive Wassermann reaction, there may be a spinal tumor. If no improvement follows arsenotherapy and mixed treatment for a few weeks, or if the symptoms advance in spite of such treatment, operative intervention is indicated. Patients with gumma of the spinal cord usually improve very rapidly after treatment, while syphilitic patients with a cord tumor will not so improve. Occasionally, it is not easy to differentiate between diffuse syphilitic disease, primary malignant bone disease, and true cord tumor.

The suspicion of *Pott's disease* has, in my experience, led to errors in diagnosis and to undue delay in not a few instances, especially on account of the changes in the vertebrae found in the roentgenograms (Chapter XIII). In the typical cases of tubercular bone disease of the spine, with evening rises of temperature, marked kyphosis, and x-ray changes, the differential diagnosis does not present any special difficulties.

In some cases, the clinical history of the patient may be very similar to that of a slowly increasing cord compression, and in the form of tubercular spondylitis which affects especially the posterior part of the bony spine (*mal vertébral postérieur* of Lannelongue), neuralgic symptoms may exist for a long time before the signs of increasing pressure upon the cord become evident.

An erroneous diagnosis of Pott's disease is more frequent than, vice versa, the diagnosis of cord tumor in a patient with tubercular bone disease.

An individual who has a tubercular spondylitis of years' duration, may develop a spinal cord tumor at or near the level of the vertebral disease. I have operated upon a case of extramedullary tumor in front of the cord, in a patient with a gibbus and a roentgenogram typical of tubercular disease at the vertebral level of the new growth. The patient, a man of thirty-two years, had had a tubercular spondylitis in early adolescence.

In rare instances an encapsulated extradural cold abscess may make increasing pressure upon the cord.

In most of the cases of Pott's disease, however, the cord symptoms progress much more rapidly than in cord tumors, and Brown-Séquard symptoms are rare.

Very rarely chronic inflammatory processes in the meninges may cause symptoms or signs which are in some respects similar to those produced by spinal cord tumor. Such difficulties in diagnoses are, however, rare. Occasionally there may be spinal cord symptoms, a subarachnoid block and an increase of protein in rare cases such as Nicholas-Favre disease.

On account of the osseous changes shown in the x-ray plates, it may be difficult to differentiate between an extradural growth that has caused erosion of bone, and tubercular bone disease; and in some cases, an exploratory operation may be necessary in order to decide the question.

Primary or secondary malignant disease of the vertebrae or soft parts may closely simulate a true spinal cord neoplasm (Chapter XIII). In most cases of malignant disease the onset of the symptoms is much more acute and the progress of the signs of spinal cord compression is very rapid; so that the period of time from the beginning of cord signs to complete spastic or flaccid paraplegia often lasts little more than a few days—frequently only twenty-four hours.

The painful symptoms are often excessively severe, and are irregular in their distribution. The patients may declare that their pain runs "straight through the body from before backward."

Malignant disease, especially when it affects the pelvic bones or the lower thoracic and lumbar vertebrae, is often associated with marked atrophy of the muscles of the thighs and legs. The pain is often so severe that movement of one or the other limb, or turning in bed, is possible only with great suffering. The distribution of the pain does not, however, correspond to any radicular or segmental area, and a careful search will often reveal a very localized bone tenderness.

In the large growths of the cauda equina, the pain in the back may be a very prominent symptom, aggravated on the slightest movement of the back. It is in this location that the differentiation between cauda tumor and malignant bone disease may be difficult. Malignant bone disease more often causes a complete flaccid paraplegia, while in caudal tumors such a complete lesion is rare.

Every patient with level signs of spinal disease, especially if there is a history of a prolonged period of pain, should be carefully examined for evidence of malignant disease in some other part of the body, to which the spinal disease may be secondary. Metastatic carcinoma of the spine is especially frequent in women with carcinoma of the breast or after the removal of a cancerous mammary gland. In women, especial attention should be paid to the breasts and the thyroid gland; in males, the prostate should be particularly examined. Metastatic carcinoma of the vertebrae may occur at any spinal level, but when secondary to prostatic carcinoma, it usually affects the lumbar vertebrae, and causes, among other symptoms, severe pain in the groins.

Bone metastases from a hypernephroma are not rare, and the vertebrae—especially in the midthoracic region—are frequently the site of a secondary growth. As this form of tumor may increase in size very slowly, the patient with spinal metastasis from a hypernephroma may suffer from root pain for a long period before spinal cord symptoms appear. In every patient with suspected vertebral disease, therefore, the kidneys should be carefully palpated and an enlarged kidney looked for on the x-ray plate.

Carcinoma of the spine is always metastatic from disease in some other part of the body, and almost regularly affects the bodies of the vertebrae, while sarcoma is either primary in the vertebrae or in the soft tissues around the spine, or is a direct extension from a tumor in the neighborhood. It often begins in or around the arches of the vertebrae. Therefore sarcoma is especially apt to form a bulging swelling on either side of the spinous processes. The back of the patient should always be carefully examined; otherwise the surgeon may discover a previously unrecognized tumor mass when the patient is in the prone or semiprone position on the operating table.

In metastatic cancerous disease of the spine, there is often a period of pain, followed by exaggeration of deep reflexes without any objective sensory disturbances; and there is not so rarely complete paralysis of the lower limbs without disturbances in cutaneous sensibility—the typical *paraplégie douloureuse des cancéreux* of Charcot. Such a clinical picture almost never occurs from compression by tumors of the cord or its membranes.

In the differentiation between malignant bone disease and spinal cord tumor, the x-ray examination is of great diagnostic value, but in many cases the x-ray plates do not show all of the bone destruction which exists. In some instances of malignant disease of the vertebrae we have been surprised at the amount of bone destruction found at operation, but not shown on the x-ray plates.

When one or more vertebrae show the "bitten out" appearance of eroded bone, especially if there are evidences of similar bone destruction in other bones, the condition is one of metastatic malignant disease. The amount of bone destruction may be so great that the bodies of several vertebrae are found to have collapsed, with narrowing of the vertebral bodies and disappearance of the intervertebral discs.

In most instances, malignant disease of the spine runs a more rapid course, especially as regards the signs and symptoms of cord lesion; and complete flaccid paraplegia with loss of all cutaneous and tendon reflexes is very frequent.

Multiple myeloma in the vertebral bodies may cause symptoms very like those of a spinal cord tumor, and in every case of suspected bone disease, the urine should be examined for Bence-Jones albumose. I have seen 5 patients with myeloma with the signs of a rapidly advancing spinal compression.

Arthritic changes in the vertebrae with new bone formation, may, in rare instances, cause root symptoms followed by level signs of cord compression. The overproduction of bone may cause pressure upon anterior and posterior nerve roots and upon the cord itself. But the differentiation between symptoms due to these spondylitic changes and those of a true cord tumor, is usually easy. In the former disease the symptoms are more diffuse and the radicular disturbances may occur over considerable areas.

When the symptoms are those of progressive spinal compression at a definite level, a moderate amount of spondylitis should not deter the physician from making a diagnosis based upon the symptoms and signs; and too much weight must not be placed upon the x-ray findings.

Benign bone tumors may sometimes cause all the signs and symptoms of a primary cord tumor. These benign growths are osteomata, chondromata which arise from the intervertebral discs, or hemangioma of a vertebral body. The large chondroma and the hemangioma can usually be recognized on the roentgen films. The small ventral chondromas (herniations of the nucleus pulposus) are discussed on pages 467 to 480. They make pressure upon the cord from before backward. Benign tumors of the vertebrae are rare.

Occasionally, *chronic inflammatory processes* in the dura cause localized thickening of that membrane, a thickening which may form an actual tumor mass with gradually increasing pressure upon the spinal cord. The pachymeningitis cervicalis hypertrophica first described by Charcot is a rare affection, of which I have seen only two examples; but a chronic inflammatory process of the dura with thickening of that membrane may occur at any level of the cord. The pachymeningitis usually involves a considerable area of the dura, and therefore a number of nerve roots. When there are pains referred to three, four, or more nerve roots, the diagnosis of pachymeningitis must be seriously considered.

Occasionally a diffuse tumor of the dura presents the clinical signs and gross appearances of a pachymeningitis. In such a patient on my service, recently operated upon, the dura over the area exposed by the operator, appeared uniformly thickened and the process was supposed to be a pachymeningitis. The histological examination of a piece of excised dura showed, however, that we were dealing with a lesion which presented all the characteristics of a diffuse endotheliomatous infiltration of the dura mater.

Much has been written on the subject of *circumscribed serous* meningitis. Oppenheim and Krause first described this affection. German authors have paid special attention to this condition and have made attempts to differentiate it from spinal tumor (Auerbach, 1920; Marburg; and others). The French call the affection *arachnoidite cloisonnée*, and Spiller, Musser and Martin (1902) speak of cysts of the spinal cord. Horsley (1909) described an affection which he called chronic spinal meningitis, but it is very doubtful whether Horsley's cases were anything more than irregular adhesions between the different membranes of the cord.

Undoubtedly, in rare instances, true leptomeningeal cysts are met with as the result of an inflammatory process with the formation of closed cavities of the arachnoid. However, many cases have been reported that were not arachnoid cysts, but only collections of fluid above an obstruction in the spinal canal.

The clinical features of extradural spinal cysts and of so-called adhesive spinal arachnoiditis are described in chapters VI and XII.

Disease within the substance of the cord may closely simulate an intramedullary tumor, and sometimes the differentiation of intramedullary disease from extramedullary compression by a new growth is not easy. We now know that the signs formerly considered diagnostic of intramedullary disease—especially a painless onset, dissociation of tactile from pain and temperature sensibility, marked muscle atrophies, fibrillary twitchings of the muscles—may occur in extramedullary cord compression. The two diseases which have to be especially considered in this connection are multiple sclerosis and spinal gliosis or syringomyelia.

The differential diagnosis may present difficulties only in the early stages of these affections. When the symptoms of multiple sclerosis are well marked—scanning speech, nystagmus, pallor of the optic discs, absent abdominal reflexes, spastic phenomena in the lower limbs without any marked sensory disturbances (especially tactile) —the disease should be recognized without difficulty. In the early stages of multiple sclerosis, however, many of the symptoms may be wanting, and then a careful study of the history of the patient and the progress of the disease is necessary. It will then usually be found that the spinal symptoms have been of longer duration and without the steady progression seen in expanding lesions. The spastic phenomena are often out of proportion to other spinal symptoms. On the other hand, the presence of nystagmus does not mean multiple sclerosis, as nystagmus may occur in high cord tumor. In multiple sclerosis, cutaneous sensory disturbances may be marked up to a distinct level but it is seldom possible to obtain a distinct level for tactile sensibility. On the other hand, disturbances in vibratory sensibility occur fairly often in sclerosis, but I have rarely found a distinct affection of joint sensibility in that disease. Examination of the spinal fluid may be helpful in making the decision between multiple sclerosis and cord tumor, for increase of protein is rare in the former disease, while the manometric tests should always be negative.

Occasionally, the differential diagnosis between gliosis or syringomyelia and cord tumor is difficult, for an extramedullary growth may present a syringomyelic symptom complex. The progress of symptoms is, however, more gradual in the intramedullary disease, and trophic disturbances of the soft and of the bony tissues are more frequent. Sensory root symptoms (root pains) are rare in gliosis, but motor root or anterior gray horn disturbances (atrophic palsies) are frequent. On the other hand, pure dissociation of cutaneous sensibilities is usual in spinal gliosis, but not as often observed in extramedullary lesions. Brown-Séquard symptoms are also rare in intramedullary disease.

Extreme grades of sensory loss over the entire body below the level are unusual in spinal gliosis unless the disease is far advanced. In the latter disease, there is often a gradual shifting upward of the segmentary level, which is unusual in extramedullary cord tumors.

It is often difficult to exclude syringomyelia in a case of intramedullary tumor, because in the early stages of the latter, the clinical picture approximates that of the former disease. When, however, there is marked compression of the cord from within, the dissociation of cutaneous sensibilities very often disappears or becomes ill defined, and sooner or later the patient presents the picture of a more or less complete transverse lesion of the cord.

It is unusual for affections other than those already mentioned to present symptoms similar to those of an expanding lesion within the spinal canal, but very occasionally, varicose veins of the spinal cord, multiple neuritis, progressive necrosis of the spinal cord (Kernohan, 1938), arteriosclerotic myelopathy (Keschner and Davison, 1933), or aneurism of the aorta will have to be considered as possibilities and may have to be excluded.

In some instances, tumors of the cervical cord begin with muscle atrophies and simulate amyotrophic lateral sclerosis or a combined system disease (Friedman, 1926; Neri, 1929; and others).

The atrophy most often begins in the intrinsic muscles of the hands, but the most marked changes may be first observed in the pectoralis major, biceps, and triceps. Although paresthesias have occasionally been described in an affected upper extremity, actual pain and objective sensory loss do not occur in amyotrophic lateral sclerosis.

Examination of the fluid obtained by lumbar puncture is always a valuable diagnostic aid, for in the majority of extramedullary (and extradural) tumors, there are characteristic changes in the pressure and in the protein content, and, in many cases, changes in the color of the cerebrospinal fluid.

Finally, especial mention must be made of the fact that the large tumors that occur over the lumbosacral cord and between the roots of the cauda equina sometimes cannot be distinguished from radiculitis of the caudal roots (*q.v.*).

REGIONAL DIAGNOSIS

In typical cord compression, there is spastic paralysis below the level of the lesion with hypertonicity of the lower limbs; or, in advanced cases, the lower limbs are in permanent flexion, or flexor spasm alternates with relaxation and extension. The sensory disturbances are usually most marked in the most peripheral dermatomes. Variations from this typical picture are frequent, however. For example, in large growths which involve the lumbosacral segments with, perhaps, the roots of the cauda equina, the paralysis may be entirely flaccid. In tumors in front of the spinal cord, the sensory disturbances may be most marked just below the level of the compression.

The signs of interference with the functions of the segments which are actually compressed, can often be distinguished from the signs referable to the cord, below the actual compression, which are due to the more or less complete isolation of the lower part of the cord and the interference with descending and ascending pathways.

The symptoms at the level of the tumor are those due to the actual pressure upon cord segments and sensory and motor roots. The pressure upon sensory roots causes pain with subjective and objective root disturbances (hyperesthesia, hypesthesia, anesthesia), while involvement of motor roots is followed by spasm of muscles, weakness or paralysis with atrophy and diminution or loss of tendon or cutaneous reflexes. The disturbances due to interference with the function of the affected segments of the cord may be difficult to distinguish from the signs referable to the motor and sensory nerve roots. The former may overlap the latter or may merge gradually into them.

1. *The Level.* For a correct level diagnosis, a careful history and physical examination are indispensable. The diagnosis of the level of the tumor usually means the determination of the uppermost segment or segments whose functions have been disturbed, and therefore what is ordinarily spoken of as the "level" of a tumor means the "upper level" of the growth.

The decision as to the upper level of the tumor can be arrived at only after a careful critical study of the highest signs of disturbed cord function—changes in power and sensation and alterations in the superficial and deep reflexes. The more careful the examination, the more exact will be the level diagnosis. In most patients, the signs and symptoms are unequivocal and the highest affected level is easily determined.

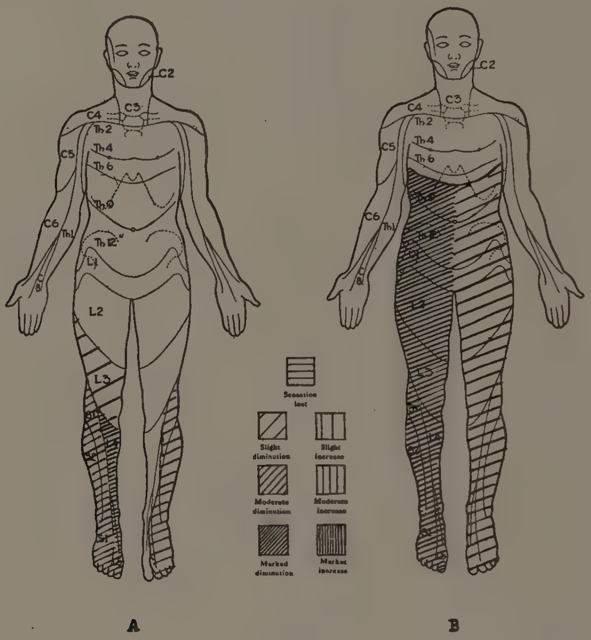

FIG. 205. Cutaneous sensory disturbances for touch and pain in a patient with a tumor at the fifth thoracic segment. A. Before lumbar puncture. B. After lumbar puncture.

In not a few instances in which the level signs were not definite, a distinct sensory level could be observed after a lumbar puncture had been performed and fluid removed (Fig. 205). I cannot emphasize too strongly the importance of an examination of reflexes and of sensation soon after the lumbar puncture has been performed. In a number of our patients, the motor and sensory disturbances of which we were uncertain became definite and positive at the examination made after the withdrawal of spinal fluid.

Not so rarely, the changes in cutaneous sensibility are very slight over the uppermost dermatomes; and unless this fact is appreciated, the tumor may be localized a number of segments below the actual level. This is especially likely to occur in tumors on the posterior aspect of the cord between the lower cervical and the middle thoracic segments.

Muscle atrophies in the upper extremities occur fairly often with tumors in the upper cervical cord, so that wasting in the forearms and hands does not of necessity signify that the tumor is at the level of the lower cervical segments.

Occasionally, there are slight sensory disturbances in dermatomes above the level of the spinal compression, and this has been explained as the result of a column of cerebrospinal fluid above the level of the tumor. Tingling and other subjective sensory disturbances sometimes occur in the upper limbs when the tumor is located in the thoracic cord below the second thoracic segment. The disturbances are supposed to be of sympathetic origin (Barré and Schrapf).

At most levels of the spinal cord, one is very apt to look for the growth a little lower than its actual location. In tumors of the conus and roots of the cauda equina, however, the tumor may be found somewhat lower than the region first exposed by the surgeon, and it is often difficult to determine whether the disturbances caused by a growth are due to a combination of cord and root signs, or to signs entirely of radicular origin. Double lumbar punctures, at two different levels, are of value for exact localization.

Babinski and Jarkowski (1912) have claimed that *the lower level of a spinal lesion* can be accurately determined by a study of the *réflexes de défense*, and that the highest muscles that take part in the defense reflexes are those supplied from the lowest segments involved in the compression. This would be a valuable method for the determination of the length of a tumor were it not for the fact that the length of the majority of cord tumors is only a few centimeters.

Injections of air or of substances impermeable to the x-ray, such as lipiodol, are much used for the determination of the level of the spinal block. Injections of air rarely give good x-ray pictures. By means of lipiodol, injected by the cisternal or lumbar route, the exact localization of a tumor can almost always be made.

2. *The Differentiation between Extradural, Extramedullary, and Intramedullary Growths.* It is often possible to determine beforehand whether a spinal tumor lies outside or inside the dural sac.

Extradural tumors may cause bone absorption which is evident in the x-ray picture. The course of the disease and the level signs of extradural growths are seldom as clean cut as those of tumors inside of that membrane, because in the former, the pressure is often more diffuse.

If there is, in addition to the growth within the spinal canal, an extension into the soft tissues, it is probable that the intraspinal growth is extradural. The condition for which Flatau and Sawicki (1922) have proposed the name "cervical neurofibroma," is one in which there is a dumbbell-shaped tumor that originates from nerve roots, one part lying within the spinal canal, and the other part forming a tumor at the root of the neck. The accompanying photographs (Figs. 100, 206, 207) are from a case of a child with such a "cervical neurofibroma," with paralyses of both upper limbs and spastic paraplegia of both lower limbs.

It is well to remember that malignant disease from the bones or soft tissues is usually extradural, so that when there is a suspicion of malignancy, the growth is probably extradural (Chapter XIII).

Differentiation between extramedullary and intramedullary growths is possible in most cases. Dissociation of the cutaneous disturbances occurs frequently in extramedullary compression, especially in cervical tumors. However, tactile sensation is rarely entirely normal. In extramedullary tumors, the cutaneous sensory disturbances are (with some exceptions in ventrally placed growths) most marked in the most caudal dermatomes, while in intramedullary growths the most marked sensory loss is usually at and just below the upper level of the lesion.

In expanding disease within the substance of the cord, there is rarely, unless much of the transverse diameter of the cord is involved, an affection of vibratory and articular sensibility.

From the standpoint of relative frequency, tumors most often lie on the posterior or posterolateral aspects of the cord. In these locations, root symptoms often occur early, and the more the growth lies to the side of the cord, the more distinct is the Brown-Séquard type of sensory and motor disturbance. If the patients are seen early in their disease, distinct posterior column disturbances (diminution in vibratory and articular sensation) may already exist. If,

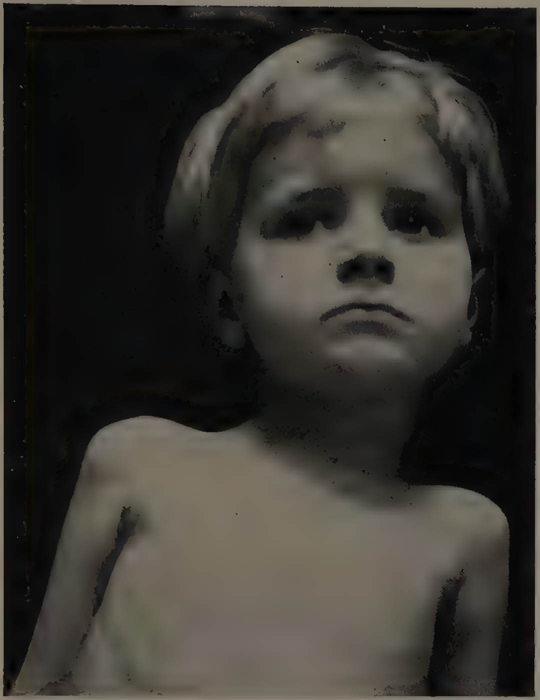

FIG. 206. Swelling on the left side of the neck in a child with a so-called cervical neurofibroma. One part of the growth (Fig. 207) lay within the spinal canal and the other part formed the tumor at the root of the neck.

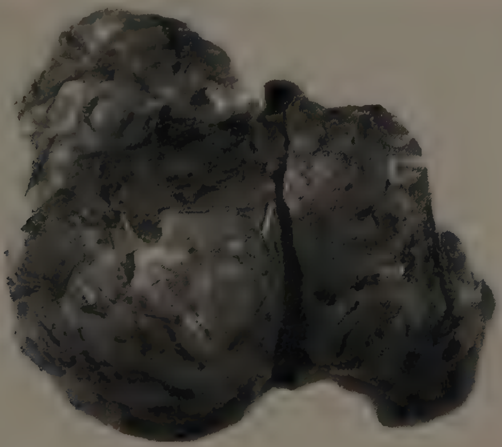

FIG. 207. The tumor removed from the patient whose photograph is shown in Figure 206. The smaller part of the tumor to the right was within the spinal canal; the greater part, to the left, was in the deep part of the neck.

however, considerable of the transverse diameter of the cord is already involved, then posterior column disturbances (changes in vibratory and articular sensibility) have little localizing value. If the signs of cord involvement are well marked without disturbances in either vibratory or articular sensibility, the growth is probably on the anterior or anterolateral aspect of the cord.

Tumors that lie on the anterior aspect of the cord to one or the other side, and those that lie on the anterolateral aspect of the cord, frequently cause paresthesias or other subjective sensory disturbances in the contralateral limbs below the level of the compression, and these contralateral subjective sensory disturbances have, therefore, considerable diagnostic significance.

Growths on the anterior aspect of the cord are often characterized by a painless onset and by early muscle atrophies, if the growth be at the lower cervical segments. Fibrillary twitchings of the atrophied muscles are not at all rare. In some of these anterior growths the cutaneous sensory disturbances are very similar to those of intramedullary growths, and the sacral dermatomes are often relatively little involved.

Errors in the diagnosis of the side of the cord on which the tumor lies may be made in those cases in which the cord is pushed to the opposite side of the canal so that the contralateral tracts of the cord are first compressed (reversed Brown-Séquard syndrome), or when a tumor pushes the cord backward against the posterior wall of the spinal canal so that posterior column disturbances, so characteristic of cord compression from behind, occur early.

CHAPTER XI

TUMORS OF THE SPINAL CORD, NERVE ROOTS, AND MEMBRANES

V. TREATMENT

OPERATIVE TREATMENT

Indications for Operative Intervention

WHENEVER THE DIAGNOSIS OF COMPRESSION OF THE SPINAL CORD BY AN EXTRAMEDULLARY NEW GROWTH HAS BEEN MADE, OPERATIVE intervention should follow without more than the necessary delay. The diagnosis can be made with certainty in many cases, and with a fair degree of certainty in the majority. Even when the diagnosis is not certain, a slowly progressive spinal affection with distinct level localizing signs, when metastatic disease has been excluded, is a case for an exploratory operation.

Surgical intervention may have to be delayed, however, on account of the general condition of the patient or because of some local disturbance, such as bed sores that have to be healed if they are in or near the field of operation.

Intramedullary growths with persistent level signs should be operated upon, not only because the differentiation between a growth inside and one outside of the cord may be uncertain, but also because some intramedullary tumors can be removed, and because the symptoms due to irremovable growths can be much improved by the decompressive effect of the laminectomy, per se, and perhaps by decompressive incision into the cord substance.

Unless there is clear evidence that an extradural tumor is a bone metastasis, an exploratory operation should be performed. Many an extradural tumor, even if it has caused absorption of bone clearly visible on the x-ray plate, can be completely removed even if it is an extension from a growth outside of the spinal canal. Not a few of these tumors are well encapsulated and relatively benign. The only cases in which operative intervention is contraindicated are those which have caused an acute flaccid paraplegia, and in which, in all probability, the spinal symptoms are not due to pressure but to interference with the vascular supply of a part of the spinal cord.

The details of the technique of the exposure of the spinal cord by laminectomy are described on pages 520 to 534. A few remarks concerning the actual removal of the tumor are made in the following pages.

As soon as the spinous processes and laminae have been removed and an extradural growth has been exposed, its extent must be investigated and, if necessary, more laminae must be taken away in order to lay bare the upper and lower ends of the growth. If the tumor surrounds the dural sac on all sides but is not adherent to the dura, it must be carefully peeled away from that membrane. Very often all neoplastic tissue cannot be removed and some of the growth on the anterior surface of the dural sac will remain behind. If the growth is found to extend beyond the confines of the vertebral canal and is encapsulated, the extravertebral part must be removed in one piece, if possible.

If, on the other hand, the growth is intradural, then the dura must be incised in a longitudinal direction without injury of the arachnoid. If a tumor is not seen, the operator must note whether cardiac and respiratory pulsations of the cord are present. The pulsation of the cord is most distinct in the cervical region and therefore an absence of pulsation is especially noticeable in that location. If cerebrospinal fluid continues to escape from above (after the arachnoid sac has been opened), there is considerable probability that the tumor is not at a higher level than the part of the cord exposed.

If the posterior spinal vessels are enlarged and tortuous, it is clear evidence that the tumor is at a higher level and is obstructing the return flow of blood in the posterior spinal veins (Fig. 208).

If the growth is not seen at the exposed level, a small probe bent at an angle must be carefully passed upward and downward in order to search for an elastic resistance to the passage of the probe. If none is found, an assistant is asked to compress the veins of the neck; if the growth is at a higher level of the cord than that exposed, the arachnoid sac will not become distended with fluid. If the tumor is at a lower level than that exposed (which is rare) there will not be any obstruction to the passage of fluid downward when the jugular veins are compressed.

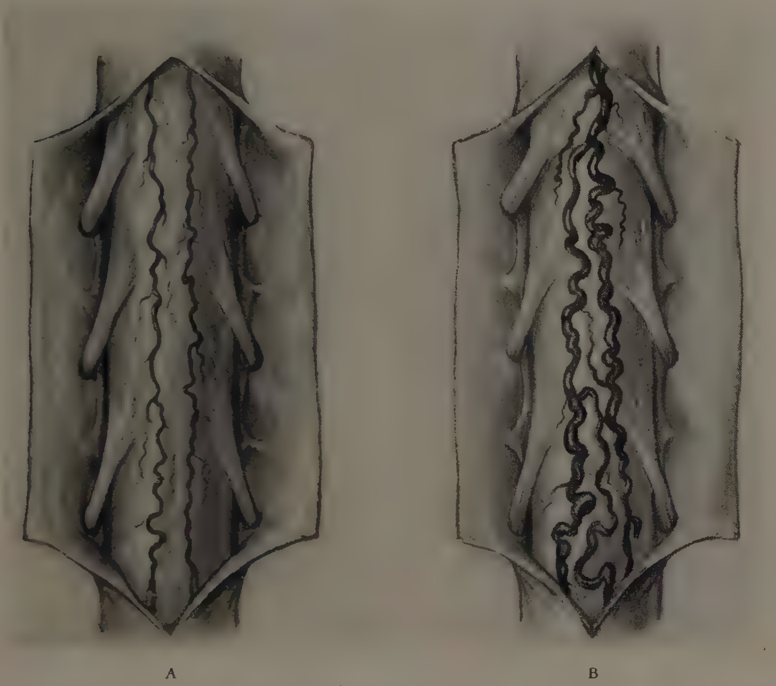

Fig. 208. A. The normal vessels on the posterior surface of the spinal cord. B. The enlarged and tortuous posterior spinal veins below the level of compression of the cord by a tumor.

If the growth lies on the dorsal or dorsolateral surface of the cord and its upper and lower poles have been brought into view, it must be carefully separated from adhesions and pulled away from the cord. Great care and gentleness must be used in all of the manipulations, and a small strip of cotton must be placed above and another below the growth in order to wall off the subarachnoid space. If the growth is encapsulated, a traction suture will often be useful, and the attempt should always be made to remove the growth in one piece (Fig. 209).

If, on the other hand, the growth is so fragile that a fragmentary removal is necessary, care must be taken that all of the capsule is removed and that fragments of tumor tissue are not left behind. Finally, the tumor bed must be inspected and the slightest bleeding controlled by muscle implants. There is seldom any difficulty with bleeding vessels if the growth has been well exposed and carefully removed. If the growth is adherent to the dura, as is often the case with the meningeal fibroblastomas, the adherent dura must be excised with the growth. If a nerve root runs into the tumor, as in the perineurial fibroblastomas, it must be divided after it has been crushed with forceps.

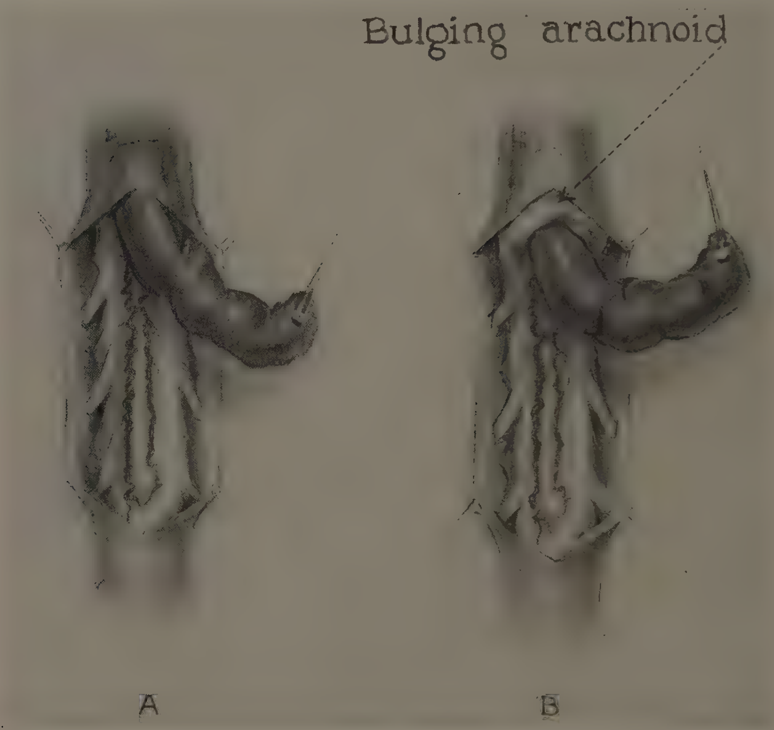

FIG. 209. A. After exposure the tumor is partly drawn out by a traction suture. B. The upper pole of the tumor is delivered by compression of the veins of the neck resulting in bulging of the arachnoid above the growth.

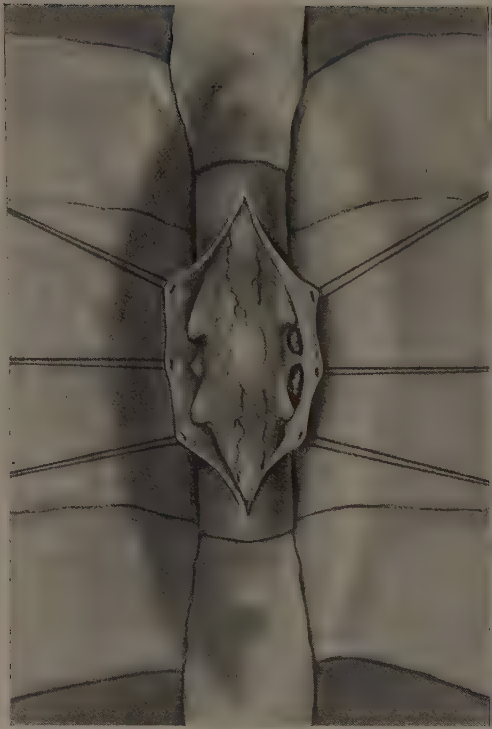

FIG. 210. The exposure of a tumor in front of the cord. The growth is seen on the right side underneath a slip of the dentate ligament.

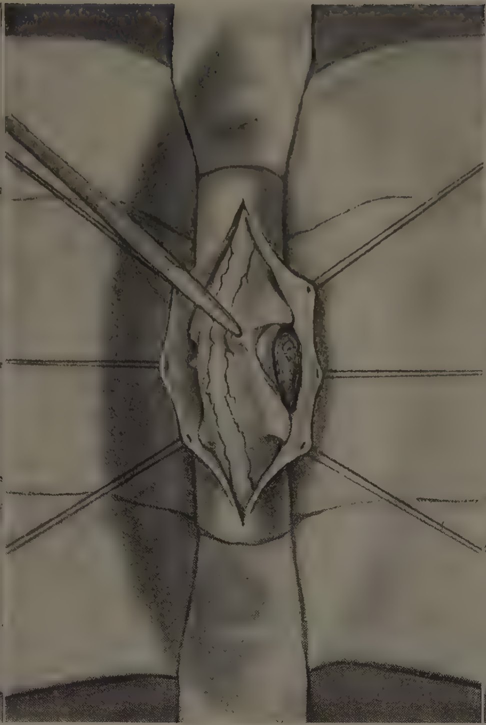

FIG. 211. The exposure of a tumor in front of the cord. A slip of the dentate ligament has been divided, and the cord raised and rotated by traction on the slip. The tumor is not yet fully exposed. (Compare with Figs. 210 and 212.)

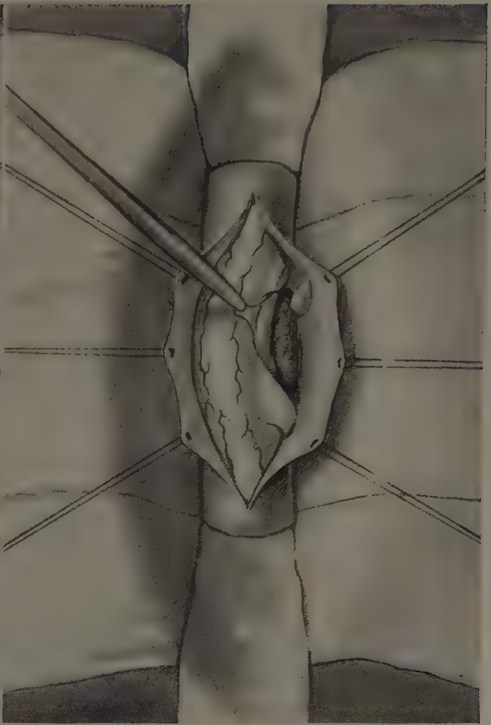

FIG. 212. The exposure of a tumor in front of the cord. The tumor further exposed after division of one posterior nerve root. (Compare with Figs. 210 and 211.)

Growths on the ventral aspect of the cord are sometimes difficult to expose until one or two slips of the dentate ligament and perhaps one nerve root have been divided. If necessary, the cord should be elevated and slightly rotated by traction on a slip of the dentate ligament. The cord should never be elevated with an instrument. Unless the growth is *very* small, it should never be drawn out from in front of the cord by possible traction. I have witnessed irremediable injury done to the cord by such a manipulation. In the case of meningiomas, the nerve root is usually so tightly stretched over the growth, that the root has to be divided. In some very soft perineurial fibroblastomas, the growth can be drawn out from under the root, especially if the latter has been stretched with two fine

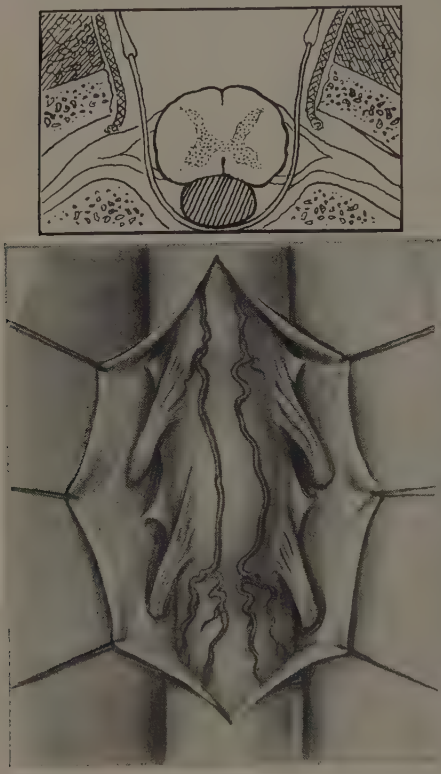

FIG. 213

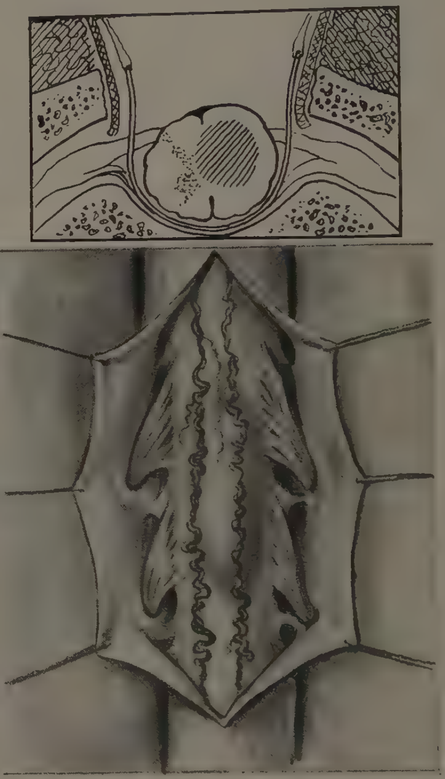

FIG. 214

FIG. 213. A ventral tumor exposed at operation. The appearance is very similar to that seen in some intramedullary growths. (Compare with Fig. 214.)
FIG. 214. An intramedullary tumor exposed at operation. (Compare with Fig. 213.)

forceps. However, even in such a case, it is usually preferable to divide the root (Figs. 210, 211, 212).

For the successful removal of an extramedullary tumor with the minimum of trauma to the spinal cord, the upper and lower limits of the growth must always be well exposed, and the surgeon should never hesitate to remove the arches of one or of several vertebrae secondarily, so as to bring the upper and lower poles of the growth into view. As soon as this has been accomplished, a narrow strip of cotton must be inserted into the subarachnoid space above and below the growth in order to prevent the passage of blood beneath the arachnoid. After a meningioma has been freed from its attachment to the dura, and in the case of a perineurial fibroblastoma, the upper pole of the growth may be delivered by having an assistant compress the jugular veins in the neck (Fig. 209 B).

Tumors that lie entirely ventral to the spinal cord may dislocate the cord backward and make it appear broader, so that the surgeon may at first suspect that he has to deal with an intramedullary growth. Therefore, the cord should never be incised until one is certain that the growth is not ventrally situated (Figs. 213, 214).

Technique for Removal of Tumors Between the Roots of the Cauda Equina

For the proper exposure of growths in the lumbosacral regions, the laminae—which are very short—must be removed well into the articular surfaces. In the large growths that are met with in this region, the dura is often very thin, and the conus is covered by large veins that are easily injured. As the lumbosacral cord is very sensitive to pressure, scrupulous care must be taken not to make any pressure upon this part of the cord or to cause any hemorrhage around it. The removal of small growths from this region or from between the roots of the cauda equina does not present any special difficulties (Fig. 215). The extirpation of the large growths (giant tumors) (Figs. 204, 216, 217) which lie on the conus and surround the roots of the cauda equina presents a different problem. These tumors are often well encapsulated in their upper part and can be easily freed from the conus, but below they often have an intimate connection with nerve roots, many of the latter perhaps passing through the growth. If the roots have been pushed to one or the other side, the tumor can be removed without difficulty; but if the growth surrounds many of the nerve roots, the separation of the

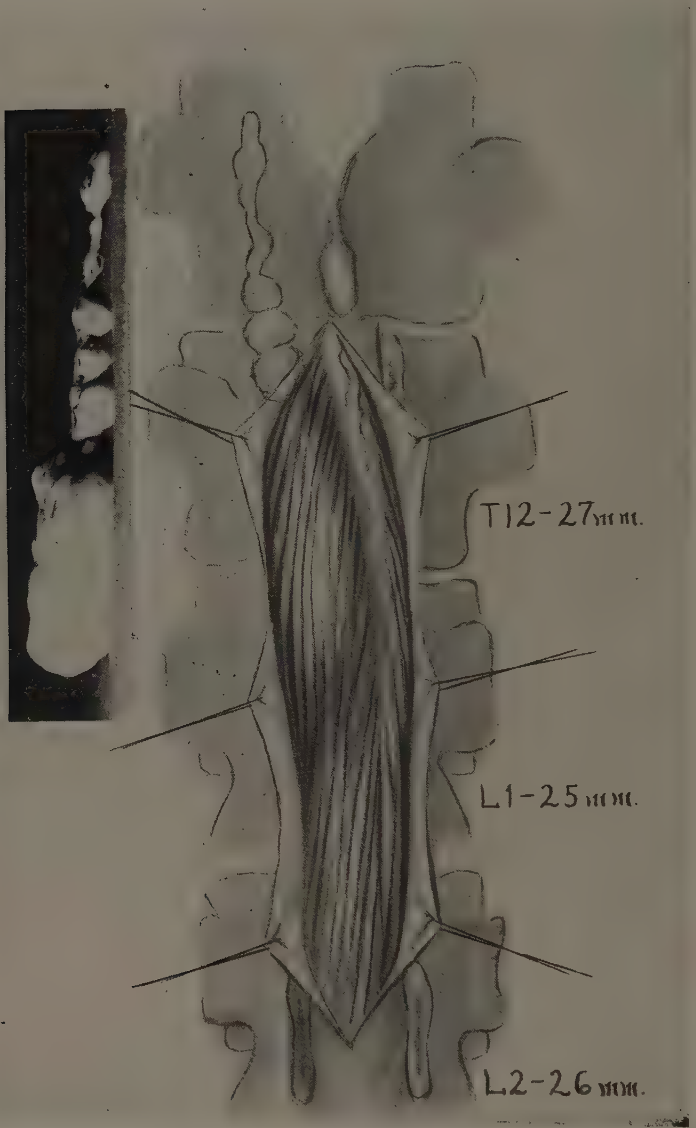

FIG. 215. A perineurial fibroblastoma under the roots of the cauda equina exposed at operation. (Insert shows size and shape of tumor.)

tumor from the roots is certain to result in severe trauma to the roots themselves. Therefore, if the connection between the tumor and a large number of the roots is a very intimate one, only as much of the growth should be removed as can be done without definite injury to a number of roots.

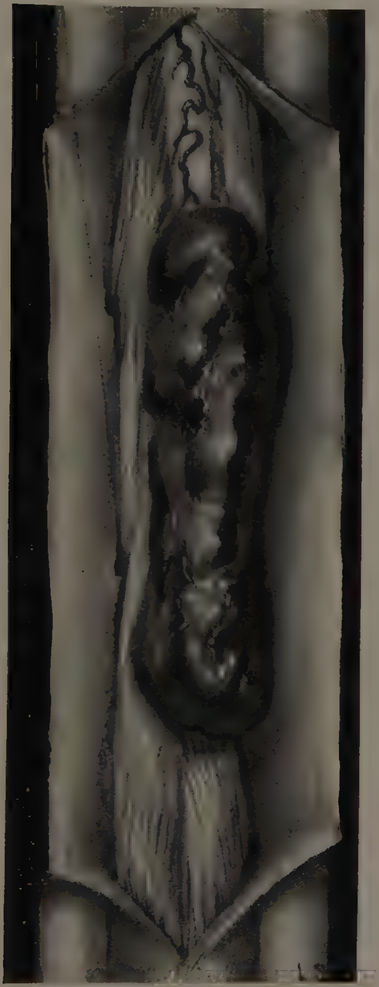

FIG. 216

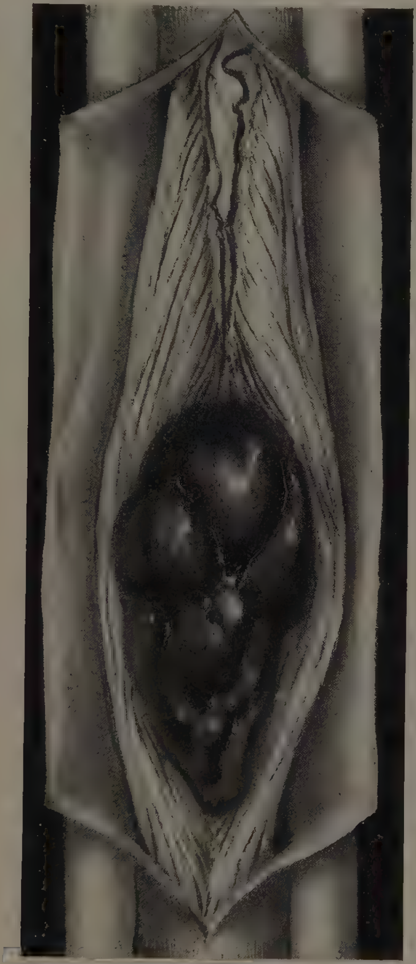

FIG. 217

FIG. 216. Giant tumor of the cauda equina exposed at operation. (Elaborated from sketch made at operation.)

FIG. 217. Giant tumor of the cauda equina exposed at operation. (Elaborated from sketch made at operation.)

Growths derived from the filum terminale can usually be well exposed by gentle traction on the caudal roots (Fig. 218). These growths are often well encapsulated and can be removed by division of the filum. In some instances, however, the growth will be found to extend into the conus, so that the lowermost part of that structure has to be sectioned.

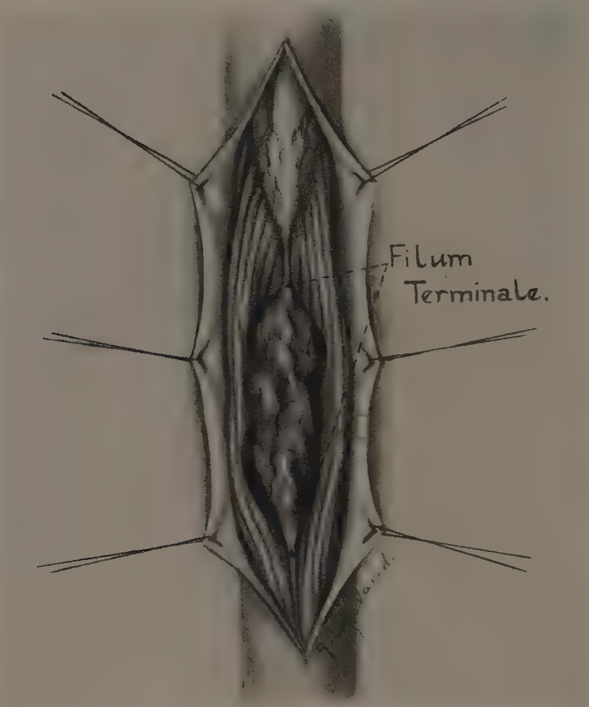

FIG. 218. Tumor of the filum terminale exposed at operation. (Elaborated from sketch made at operation.)

These large tumors often bulge very markedly as soon as the thin and very tense dura has been incised; if this is the case, it may be of advantage to conclude the operation for the time being, and to remove the tumor at a second operation, performed one to two weeks later. By that time the growth may have freed itself from many of the nerve roots.

With most of these large tumors, the removal has to be a fragmentary one. As most of them are not vascular, there is little or no bleeding when the tumor is removed piecemeal. If the growth cannot be entirely removed, the patient should have x-ray treatment as soon as the wound is healed.

In these large tumors, which frequently fill up the entire lower end of the spinal canal, the dura is often so much thinned that the cut edges cannot be sewn together again at the close of the operation.

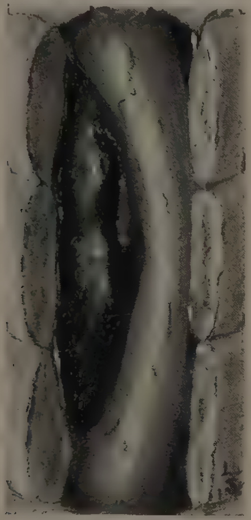

FIG. 219. A localized extradural tumor (sarcoma) exposed at operation. The growth extended around the anterior aspect of the dura. (Elaborated from the surgeon's sketch.)

In these patients, the exposed conus and cauda are covered by a piece of Cargile membrane the edges of which may be sutured to the edges of the dura so as to form a new covering for the cord.

TECHNIQUE FOR THE REMOVAL OF EXTRADURAL TUMORS

Small well-encapsulated extradural growths on the posterior or lateral aspects of the dural sac can be removed without difficulty. Many extradural growths extend around the dural sac to its anterior surface, and for the removal of these growths extensive removal of bone may be necessary, and even one or several nerve roots may need to be divided before the dural sac can be pulled away sufficiently to allow of the extirpation of the growth (Figs. 219, 220, 221, 222).

The growth may extend all around the dural sac, and it may be necessary to approach it from both sides.

A growth in front of the dura may push the dural sac markedly backward so that it almost forms a knuckle when the laminae have been removed. For the exposure and removal of these tumors, the maximum of room must be obtained, and it is often advisable to make a small incision in the dura and arachnoid and to empty the fluid in the subarachnoid space.

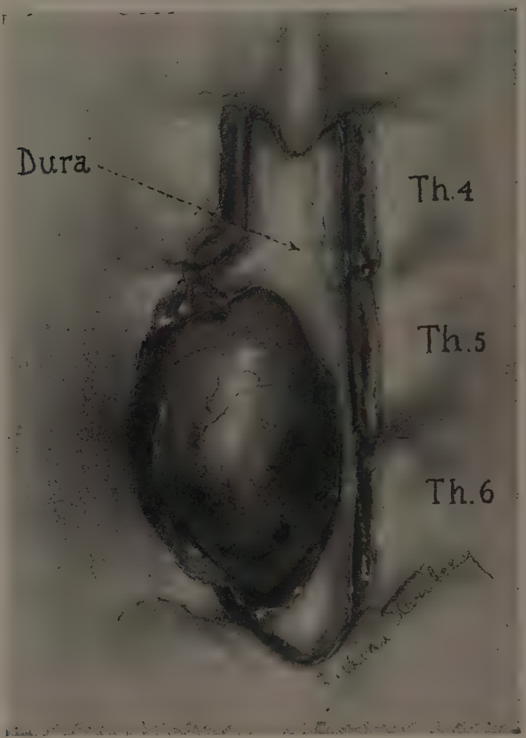

FIG. 220. A localized extradural tumor (sarcoma) which could only be extirpated after extensive removal of the vertebral arches on the left side. (Elaborated from the surgeon's sketch.)

A great deal of bone may need to be removed in order to expose the most anterior part of the growth which may be adherent to, or may have caused absorption of part of a transverse process, or of the posterior and lateral portion of a body of a vertebra. There are some well-encapsulated growths (usually giant-cell sarcomas and therefore relatively benign) that extend by a narrow pedicle into the chest between ribs or in front of a rib; and these growths can be entirely removed. There should be no hesitation in the resection of parts of one or more ribs in order to expose a part of a well-encapsulated growth that may lie between or in front of the ribs.

Benign extradural growths that have originated from one or several nerve roots outside of the dura, or even outside of the spinal canal (cervical neurofibroma of Flatau and Sawicki) may form a secondary swelling at the root of the neck. In these cases, especially if the patient is a child, the operation may have to be divided into two stages. At the first operation, the extradural mass which compresses the dural sac is removed; at the second, the large growth in the neck is excised.

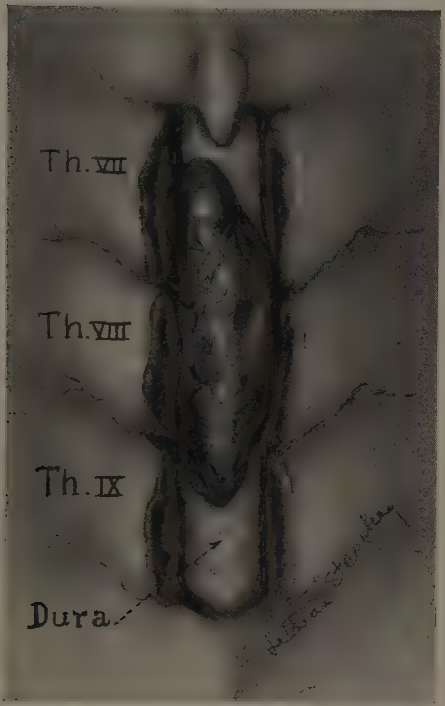

FIG. 221. A localized extradural tumor (meningioma) exposed at operation. (Elaborated from the surgeon's sketch.)

If a growth is found to be not encapsulated and if it infiltrates the surrounding soft tissues and the body of a vertebra, complete removal is impossible. The partial removal may be followed, however, by a marked improvement in the signs of spinal compression.

The exposure of some benign tumors (osteoma, chondroma) which originate from the posterior surface of the body of a vertebra may be very difficult without dislocating the dural sac to such an extent that the cord within the dura could hardly escape injury.

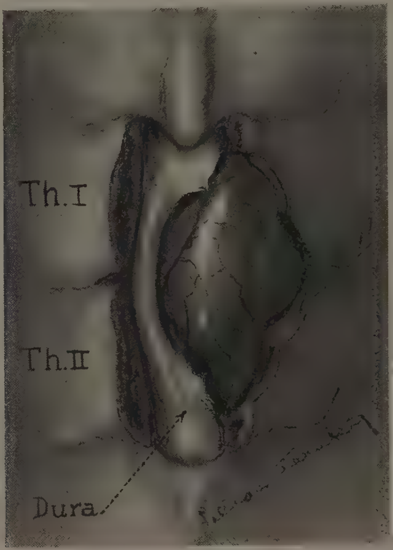

FIG. 222. An extradural tumor which could be extirpated only after removal of parts of several ribs. (Elaborated from the surgeon's sketch.)

In these cases, it is advisable to perform a transdural operation. The dural sac is first incised, and the cord then dislocated by traction on a divided slip of the dentate ligament, perhaps after section of one posterior spinal root. A second incision is then made over the dura in front of the cord (Fig. 232) and the tumor removed. In the appropriate cases, the advantages of this method are not only a better exposure of the growth, but also a diminished danger of trauma to the cord, because it is exposed and is in plain view during all the manipulations incident upon the removal of the growth.

In all surgical procedures in which the posterior surface of the body of a vertebra is exposed, there is likely to be troublesome bleeding from the venous plexuses which lie on the posterior surface of the bodies of the vertebra. The operator may be obliged to pack the wound and wait until the oozing has ceased.

Technique for the Removal of Intramedullary Tumors

The removal of intramedullary growths at the fourth cervical segment should never be attempted on account of the danger from paralysis of the phrenic nerves. Likewise, the incision of the lumbosacral cord for the removal of an intramedullary growth is rarely permissible on account of the danger of permanent loss of control of the bladder and rectum. The development of intramedullary tumor surgery began with isolated attempts to remove localized growths from the substance of the cord by incision of the cord and peeling out of the growth from the cord tissues. The first attempts were fraught with disastrous consequences, and if the patient did not have a complete transverse cord lesion before the operation, he was certain to have it afterward. In a few cases, the operator made an incision in the cord without making any attempt to remove the growth. In a patient of Cushing's (1910), marked improvement followed such a procedure, and improvement after the decompressive incision was noted also by Feiling (1920). The same result was noted in some of my patients.

In isolated instances the removal of intramedullary tumors was followed by a modicum of success, and finally more or less successful cases were reported from a number of clinics (Krause, 1906; Braun, 1906; Veraguth and Brun, 1910; Elsberg and Beer, 1911; Röpke, 1911; Schultze, 1912; von Eiselsberg, 1913; Potel and Veaudeau, 1913; Elsberg, 1914; Foerster, 1917; and others).

With the object of reducing the trauma of the cord to the minimum, I have devised the method of "extrusion" of intramedullary tumors. This method is based upon the principle that if there is an increase of pressure within the cord substance due to an intramedullary growth, an incision of the cord will put the cord in the most favorable condition for a readjustment of pressure conditions. In this process, an intramedullary growth, if it is localized, will be "extruded" and can then be removed at a second-stage operation (Fig. 223). If the growth is infiltrating, it may be partially extruded. Successful removal of intramedullary growths by the method of extrusion has been reported by Dercum and Da Costa (1916), Foerster (1917), ourselves, and others.

The appearance of the swollen cord due to a growth within its substance is sometimes very similar to a cord broadened and pushed backward by a tumor on its anterior aspect (Figs. 213, 214). In several ventral tumors of my series, the cord, when first exposed, was apparently enlarged and swollen as if from an intramedullary growth.

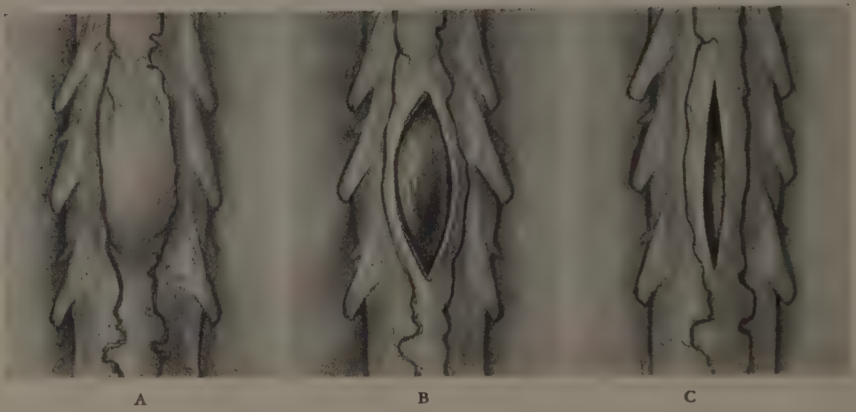

FIG. 223. Three stages in the exposure and partial removal of an intramedullary tumor. A. Tumor exposed. B. The tumor has been partly extruded. C. The extruded portion has been removed. (Diagrammatic.)

The enlargement of the cord, due to a tumor within its substance, is usually fusiform and extends over several segments. When the operator has made certain that the disease is intramedullary, he must first determine by aspiration with a fine hypodermic needle whether the swelling is due to fluid. The puncture should be made near the posterior median septum.

If no fluid is obtained, an incision ½ cm. long is made in the cord 1 or 2 mm. to one side of the posterior median septum, so as to avoid the blood vessels that run in the septum. The incision is very slowly and carefully made with a fine "von Graefe" knife. As soon as tumor tissue is exposed, it will protrude through the incision, and if it be watched for a few minutes, it will be found to be more and more extruded from the cord. No attempt should be made to remove the growth; at most, a small bit of tissue should be excised for pathological examination. The cord is covered by a piece of Cargile membrane, the dura left open, and the muscles, fascia and skin closed in the usual manner. One week later, the wound is reopened. If the tumor has been a localized one, it will have been almost entirely extruded from the cord and can be removed with little difficulty. Great care must be taken, however, in freeing the tumor from the cord; if it still be adherent, or if it is found to be an infiltrating growth a part of which has been extruded, the extruded portion is carefully excised and the remaining part of the growth left undisturbed.

If, on the other hand, very little of the tumor has been extruded it is advisable to do nothing to it. Unless the tumor was a localized and encapsulated one which had been entirely removed, the dura should not be closed, or should be sutured only in part, the opening being covered by a piece of Cargile membrane. The muscles, fascia and skin are then closed in the usual manner.

If carefully and correctly performed, no symptoms follow the incision in the cord, but if bleeding occur into the substance of the cord, a complete transverse cord lesion may be the unfortunate result of the procedure.

This is the two-stage method of "extrusion" of intramedullary tumors, described by Dr. Beer and myself. I believe that in most instances it is the only safe method for the complete or partial removal of an intramedullary tumor. This statement is made in spite of the fact that a few cases of successful removal of intramedullary growths at one sitting have been reported.

Decompressive Laminectomy

In irremovable intradural or extradural tumors, the laminectomy itself may so alter pressure conditions as considerably to relieve the cord compression. For decompressive purposes, more of the laminae should be removed than when the relief of pressure has been accomplished by the removal of the tumor. If the tumor is intradural, the dural incision must not be sutured, but the exposed cord must be covered by Cargile membrane. If the tumor be an intramedullary one, no matter whether or not an incision in the cord has been made, the dural sac must not be sutured.

As a palliative operation for the relief of severe pain, the division of the anterolateral tracts may be performed, but this operation is practically never required in true cord tumors. It is an operation that has its field of usefulness in irremediable malignant bone disease and in other painful conditions.

X-RAY AND RADIUM THERAPY

In irremovable extradural and intramedullary tumors x-ray therapy should be given. In a number of my cases, the progress of the disease has apparently been checked. Some of the patients have lived for many years after repeated roentgen treatments. Especially beneficial results have been obtained in extradural sarcoma, in Hodgkin's disease with extradural masses, and in the spinal metastases of posterior fossa medulloblastomas. In the present status of x-ray and radium therapy, it is difficult to make any positive statements, but the results thus far cannot be called brilliant. Perhaps in the future, better results will be obtained.

There are many reports of the value of x-ray and radium therapy in spinal diseases and in tumors of the spinal cord, but notes of warning have been sounded against too intensive a treatment. I have seen one patient with an easily removable extradural tumor, who had been subjected to radiotherapy before he came to us, in whom a complete loss of power in the limbs followed quickly upon the treatment.

Roentgenotherapy is therefore not a single-edged sword. It may be tried in irremovable cord tumors, and it may palliate, but it will not cure.

OPERATIVE MORTALITY

When an individual or his family is informed that the patient has a spinal cord tumor for which operative intervention is necessary, the questions usually asked are: What are the dangers of the operation? What is the outlook if the tumor is completely removed?

Will complete recovery follow and how much time will elapse before improvement will occur?

In order to answer such queries, a number of factors must be considered.

The danger attendant upon a laminectomy and removal of an extramedullary tumor is not great. If the general condition of the patient is satisfactory and the cord compression is not too far advanced, the chances for recovery from the operation are very good.

The older mortality figures were very high, but the mortality statistics of recent years are low.

The operative mortality of my cases is summed up in Table XI.

TABLE XI

OPERATIVE MORTALITY IN 275 CASES OF VERIFIED SPINAL CORD TUMOR

Old Series: Up to 1924

	Number	*Recovered*	*Died*	*Operative Mortality (Per Cent)*
Extramedullary	55	51	4	7.3
Conus and cauda	12	9	3	25.0
Extradural	20	19	1	5.0
Intramedullary	13	11	2	15.0
Totals	100	90	10	10.0
New Series: 1924–1938				
Extramedullary	83	82	1	1.2
Conus and cauda	18	17	1	5.5
Extradural	68	65	3	4.4
Intramedullary	6	5	1	16.6
Totals	175	169	6	3.4
Totals of all tumors, to 1938	275	259	16	5.8

Of the 138 patients with extramedullary tumors, 5 or 3.6 per cent died from the operation.

Many of the large cauda equina tumors cannot be entirely removed, and the fatalities from the operation are naturally greater.

Of 30 patients, 4 died, giving 7.5 per cent as the mortality of the operations in these patients.

There were 88 patients with extradural tumors, of whom 4 or 2.2 per cent died after the operation.

Operations were performed for 19 intramedullary tumors, with a mortality of 16 per cent.

A critical study of the histories of the fatalities in my series shows that most of the deaths occurred either in patients with inoperable and irremovable tumors, or from complications that were only indirectly connected with the operative procedure.

PROGNOSIS AND IMMEDIATE AND FINAL RESULTS

Table XII gives the number of patients in whom the tumor could be entirely or only partially removed or was irremovable:

Table XII

REMOVABILITY OF TUMORS

	Extramedullary	*Extradural*	*Intramedullary*	*Total*	*Per Cent*
Complete removal	150	63	7	220	80.0
Partial removal	17	11	11	53	19.0
Irremovable	1	0	1	2	1.0

Obviously, the prognosis after an operation for a spinal cord tumor will depend upon whether the growth was completely or partly removed, or was irremovable, and upon the stage of the disease in which the patients are operated upon.

A patient with an irremovable growth may improve somewhat from the decompressive effect of the operation, but sooner or later the symptoms will again become aggravated. This improvement may last for one or for a number of years, and may attain such a degree that the patient can return to his work and again become, for the period of his improvement, a useful member of his community.

Partial removal of a tumor which cannot be completely eradicated may also be followed by satisfactory results. For example, a patient with a "giant" tumor of the cauda equina which was not entirely removed, remained alive for more than thirty years, during which time she bore three children and attended to her housework.

If a patient with a removable tumor comes to operation before the compression of the cord has been of such long duration that the lesion of the cord is far advanced or is practically a complete transverse one, there is an excellent chance for the recovery to be complete or almost complete. The degree of recovery, however, will depend upon the character of the tumor; the small hard tumors cause irremediable damage to the spinal cord more rapidly than the soft (and often larger) ones. Thus, there was very little functionally useful improvement in a patient with a hard fibroma, in whom the symptoms were of only ten months' duration; and in two other patients, in whom the history of the disease dated back two years. There was, however, functionally useful improvement in a patient in whom the disease had lasted for five years.

The longest period of spinal tumor symptoms before operation, followed by complete recovery, was three years; but in the majority of instances it was from one to two years. Thus, of the patients from whom extramedullary tumors were removed followed by complete recovery, 6 per cent gave a history dating back three years, 9 per cent, of less than one year, and 85 per cent, of from one to two years.

In extradural tumors that can be completely removed, the outlook for recovery is just as good as in the intradural growths, although there may be a recurrence of the disease after a number of years. There was recurrence after nine years in one of my patients.

A better idea of the outlook for recovery can be gained if one considers not the duration of the disease from the first symptoms but the duration of the paralysis.

A spastic paralysis of one or both lower limbs, even of many months' duration, can be completely recovered from in a few weeks or months, and it is often surprising how rapidly the spasticity will disappear and voluntary power be regained in limbs that have been very stiff and without any motor power for a number of months.

If the spastic paraplegia has lasted more than two years, however, complete recovery is very rare, and considerable spasticity is apt to remain.

The return of power in the lower limbs is often more complete than it is in an upper extremity, if the loss of power in the upper limb has been marked, especially if it was associated with advanced muscular atrophies. Even then, very great improvement and sometimes complete functional recovery may occur.

In not a few patients who consider themselves entirely well, a neurological examination years after the removal of the tumor will show that the reflexes in one of the lower limbs are still hyperactive and that an ankle clonus persists.

Very rarely a patient is seen who has a flaccid paraplegia as the result of a long-standing tumor compression of the cord. In these cases, the outlook for improvement, even after a satisfactory removal of the tumor, is small.

It is never possible to say with certainty, from the appearance of the cord at the time of the operation, how much improvement will take place. I have seen a cord in which there was a great return of function, which had appeared so much flattened that much improvement after the operation seemed impossible. One must, therefore, judge more from the physical signs and their duration than from the gross appearance of the cord at the level of the spinal compression.

The duration of time that will elapse before marked improvement or complete recovery will occur, varies in different patients. Often, as has already been mentioned, the improvement begins within a few hours of the operation and progresses rapidly, so that a patient bedridden for several months from a spastic paraplegia will be able to walk within a few weeks and will recover entirely in two to four months.

In other patients, the beginning of improvement is delayed for weeks, and perhaps months. In one of my cases, improvement began only after several years. When improvement has once begun, the progress continues steadily.

CHAPTER XII

TUMORS OF THE SPINAL CORD

VI. CYSTS. HERNIATION OF NUCLEUS PULPOSUS. GUMMA

EXTRADURAL AND INTRADURAL CYSTS

THE MAJORITY OF EXTRADURAL CYSTS THAT HAVE BEEN RECORDED IN MEDICAL LITERATURE WERE EITHER DERMOIDS OF WERE DUE TO the echinococcus; they were seldom of large size, and were usually found in adults.

There is, however, a variety of extradural cyst, not dermoid nor parasitic, which occurs in adolescents and which is usually of large size. A typical case follows:

N.I. No. 16150, L.G., a boy fifteen years of age, was admitted to the Neurological Institute on May 25, 1933.

Except for diseases of childhood, the patient had always been in good health until the present illness began. He had played football during the autumn season. For about three months he had had some weakness and numbness of the right lower extremity. This had gradually become worse and had soon involved both lower limbs so that walking had become difficult. He never had pain or bladder disturbances. On account of a supposed sprained tendon around the right hip he had been kept in bed for two weeks prior to his admission to the Neurological Institute.

The patient was a well developed, well nourished, rather fat boy. He was unsteady on his feet and had a spastic gait. There was weakness and spasticity of the lower limbs, more marked on the right, with bilateral suprapatellar, patellar, and ankle clonus, Babinski and Chaddock, and foot-drop on the right side. There was no atrophy of the muscles of the limbs. Power and reflexes of the upper extremities were normal.

Tactile, pain, and temperature sensibilities were very slightly diminished below the level of the seventh thoracic dermatome. Both sides of the body were equally affected, and the exact upper level of the sensory disturbances was indistinct. There was some impairment of vibratory and muscle-joint-tendon sensibility in the right large toe. Except for slight nystagmoid jerks of the eyeballs on extreme lateral gaze, the functions of the cranial nerves were not disturbed.

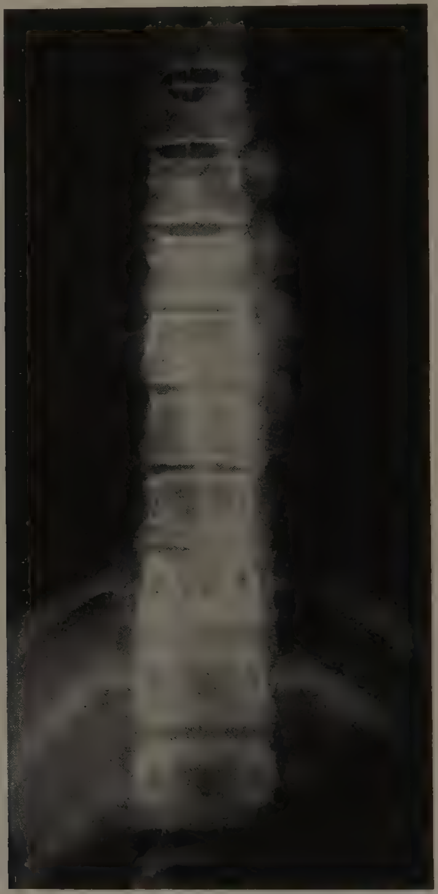

FIG. 224. X-ray showing enlargement of the interpediculate spaces of the seventh, eighth, and ninth thoracic vertebrae in a case of extradural cyst. Note the flattening and atrophy of the pedicles of the affected vertebrae.

The manometric tests by the nitrite of amyl and jugular compression methods showed that there was a complete spinal subarachnoid block. The spinal fluid contain 2+ globulin and 171 mg. of protein to 100 cc. The Wassermann test was negative.

The x-ray examination showed that the pedicles of the seventh, eighth and ninth thoracic vertebrae were definitely narrowed and atrophied, especially on the right side, and that the interpediculate space of the seventh, eighth and ninth thoracic vertebrae was much enlarged (Fig. 224).

The marked enlargement of the vertebral canal from the seventh to the ninth thoracic vertebra made it clear that the disease had existed longer than three months, and in conjunction with the obesity of the patient made one think of a congenital lesion.

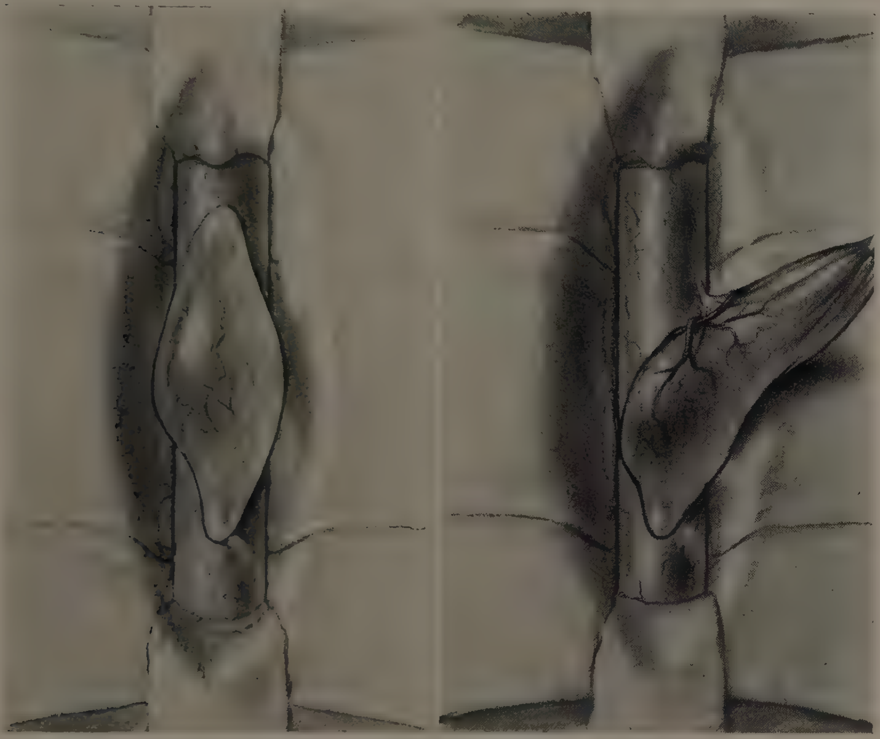
FIG. 225 FIG. 226

FIG. 225. A large extradural cyst exposed at operation.

FIG. 226. Showing the attachment of the cyst in the neighborhood of the right seventh thoracic root.

After the first examination and before the manometric tests had been made, a diagnosis of atypical multiple sclerosis or other form of intramedullary disease had been seriously considered. The existence of a complete spinal subarachnoid block with increase of globulin and protein in the spinal fluid made multiple sclerosis improbable.

The patient came under our observation at the period when a special study was being made of the pedicles and the size of the interpedicular space in the normal spine and in tumors of the spinal cord, and we were impressed by the x-ray changes.

At operation, the spines and laminae of the seventh and eighth thoracic vertebrae were first removed. A large thin-walled cyst distended by clear fluid was at once exposed (Fig. 225). In order to lay bare the upper and lower poles of the cyst, the sixth and ninth vertebral arches were then resected. The cyst was gradually freed from the dural sac underneath. During the manipulations a small tear in the cyst wall occurred and considerable clear fluid escaped. The cyst was not adherent to the dura except in the region of the right seventh thoracic root where it was firmly attached (Fig. 226). After several large vessels in this region had been ligated, the narrow attachment was divided without opening the dura. The sac was then removed in one piece. The vertebral canal was much enlarged, and the nerve roots in their sheaths were exposed for at least 1 cm. on both sides.

An examination made as soon as the patient had awakened from the anesthesia showed that there was a remarkable return of power and diminution of spasticity in the lower extremities.

Convalescence from the operation was uneventful. A manometric test was done after the wound was healed and showed that the spinal subarachnoid space was not obstructed. The spinal fluid removed at this time contained no globulin and 35 mg. of protein.

When the patient was discharged from the hospital three weeks after the operation, the strength of the lower limbs was very good, the spasticity was slight, and the tendon reflexes were only moderately overactive. The abdominal and cremasteric reflexes had returned. The patient walked without difficulty although his gait was still somewhat awkward. Sensation was normal over the body and lower limbs. One month later, his gait was normal, the tendon reflexes in the lower limbs were not hyperactive and the Babinski reflexes had disappeared.

The cyst was about 7 cm. long and 4.5 cm. wide. The walls were thin and translucent. Microscopic examination showed that the cyst wall consisted of fibrous tissue with an endothelial lining.

This history is typical for a case of extradural spinal cyst, of which about 12 cases have been reported in neurological and neurosurgical literature. The cysts are the result of herniation of the arachnoid through the dural opening for a nerve root, and in a sense they are arachnoid cysts. The sac may be both extradural and intradural or entirely intradural. In the 5 cases of my personal experience, 3 were extradural, 1 was both extradural and intradural, and 1 was entirely intradural. In the latter case, the sac lay outside of the general arachnoid membrane and was firmly adherent to the dura near the dural opening for a nerve root.

These cysts are probably due to congenital abnormalities in the development of the spinal membranes. They are most often encountered between the fourth and the tenth thoracic vertebrae, but Cloward and Bucy (1937) have reported 2 cases in the thoracolumbar region. The cyst is usually of large size and distended by clear fluid. The attachment is always near the dural opening for a nerve root and the neck of the sac is either closed off from the general subarachnoid space or communicates with the latter.

In the beginning, the diverticulum or cyst is small and does not produce compression of the spinal cord. As the cyst gradually increases in size, the dura and cord are more and more pressed upon.

The patients are most often adolescent males between the twelfth and sixteenth year of age, although 2 cases in adult men and 2 in adolescent females have been reported. The duration of symptoms may be anywhere from three months to three or more years. In my patients, symptoms had existed for three months, nine months, two years and three and one-half years.

Pain is not complained of at the beginning and it is seldom a prominent symptom. There is usually a slowly or more rapidly increasing spasticity of the lower limbs with diminution in power of the two limbs or of one after the other. There may be a fluctuation in symptoms, amelioration occurring after rest and hyperextension of the spine. The temporary improvement, which is never complete, is probably due to temporary emptying of a cyst in which there is a free communication with the subarachnoid space.

The sensory symptoms may be very slight, so that the occurrence of motor disturbances alone makes one think of an intramedullary disease of the spinal cord. In one of my patients, tactile sensibility was involved less than pain and temperature sensibilities; in another one, no evidence of tactile disturbance was discovered, while there was a definite diminution of pain and temperature senses. Vibratory and muscle-joint sensibility were diminished in all of my patients. The upper level of disturbances of cutaneous sensibility was indistinct. The vague upper level of sensory disturbances is no doubt due to the elastic compression of the dura and spinal cord by the soft fluid-containing sac.

The motor disturbances most often found are marked loss of power, spasticity and hyperactive tendon reflexes in the lower extremities with bilateral ankle clonus and Babinski reflex. The limb on the same side as that on which the cyst is attached to the dura is usually first affected, but very soon weakness and spasticity affect both lower limbs.

There is often a rounded thoracic kyphosis which may be very marked and which may become worse after the removal of the cyst (Cloward and Bucy). In most of our patients and in the cases reported by others, there was a complete spinal subarachnoid block, and the fluid obtained by lumbar puncture contained an increase of total protein (from 80 to 200 mg.) and an increase of globulin. In one case (Cloward and Bucy, 1937) there was no subarachnoid block and the protein in the spinal fluid was within normal limits.

The changes seen on x-ray films of the affected portion of the spine are characteristic. The pedicles are greatly thinned and atrophied and the interpediculate spaces of the affected vertebrae are much enlarged (Figs. 66, 67). With these changes there is often associated a kyphosis with changes in the vertebral bodies. The enlargement of the vertebral canal found on the roentgen films of a patient with a large extradural cyst is similar to that found in congenital extradural and intradural lipomas and also like that found in large tumors of the conus and cauda equina.

The congenital lipomas occur most often in the cervical region and the giant tumors in the lumbar region, while these large extradural cysts have usually been found in the thoracic region of the spinal cord.

From all of these facts one can conclude that there is a characteristic syndrome of compression of the spinal cord by a large extradural or intradural cyst.

The individual is an adolescent with the history and symptoms of a progressive spastic paraplegia. Pain is absent or is not a prominent *symptom. The objective disturbances of sensibility are slight and their upper level is in the midthoracic region, usually at the sixth or seventh thoracic dermatome. The manometric tests demonstrate a subarachnoid block with the characteristic spinal fluid changes of cord compression. Measurements on anteroposterior x-ray films show that the interpedicular spaces of three or more vertebrae are enlarged. The pedicles of the affected vertebrae are narrowed and atrophic.*

This combination of symptoms and signs with the characteristic changes in the bony spine justify the diagnosis of extradural cyst of the spinal cord.

Operative Treatment

The operative treatment consists of laminectomy and extirpation of the cyst. If the cyst is extradural, it will be exposed as soon as the first vertebral arch has been removed. The cyst wall is usually thin and translucent and must not be mistaken for a distended dura. If the upper and lower poles of the cyst have been exposed, it is a simple matter to separate it from the outer surface of the dura as it is hardly adherent excepting at the small area of attachment which will always be found at or near the exit from the dura of a nerve root. There may be one or several small vessels at the point of attachment which have to be tied or clipped before the attachment can be divided. It is usually advisable to ligate the neck of the sac with fine silk so as to prevent leakage of cerebrospinal fluid.

If the cyst is intradural, it will be found as soon as the dura has been incised. It can usually be removed without any special difficulty.

The results of the operative removal are usually very satisfactory. Unless the signs of compression of the spinal cord are far advanced, complete recovery may be expected.

HERNIATION OF THE NUCLEUS PULPOSUS (VENTRAL CHONDROMA, ECCHONDROSIS)

Large cartilaginous growths derived from the bony parts of the vertebral column, which cause symptoms from pressure upon the spinal cord and nerve roots, are not rare, and many cases of this kind have been reported in medical literature. The tumors are often of considerable size, and they may form a visible prominence on the neck or back. Their recognition is not difficult, the diagnosis can usually be made from the x-ray films, and the removal of the growths, although often time-consuming, presents no special problems.

There is another type of cartilaginous growth due in most, if not all, instances, to herniation of a nucleus pulposus of an intervertebral disc. The tumors are small in size, they are situated in front of the dural sac, their clinical course is in many respects peculiar, their localization presents some of the problems met with in tumors of the spinal cord, and their differentiation from spinal new growths may be difficult. They usually have their origin from the posterior border of a vertebral disc and from their very beginning they occupy space within the vertebral canal. Up to a comparatively recent period, this particular type of lesion had not been described, and it required almost a decade before sufficient knowledge had been gained to discover what is especially characteristic about these tumors, to acquire the necessary experience to recognize them from their clinical features, and to arrive at a conclusion regarding the best procedures for the surgical approach to them and for their removal.

As is so often the case in operative surgery, the first example of this type of lesion was stumbled upon in an exploration for a tumor on the anterior aspect of the spinal cord in which a vertebral malignancy had been suspected. I operated upon the patient in 1922, at a period when modern manometric tests and the methods for the quantitative estimation of the total protein in the spinal fluid had not yet been devised. Looking backward, it was a fortuitous circumstance that the small ventral growth was discovered at all, and that with the lack of experience in the handling of growths in that location, no damage was done to the spinal cord during the removal.

The small masses derived from the posterior surface of an intervertebral cartilage were first believed to be true chondromas or ecchondroses, but further studies have made it highly probable that they are herniations of the nucleus pulposus, often produced by trauma to the back.

Occasionally, in treatises on pathology and in publications in medical journals on the subject of cartilaginous tumors, mention was made of the fact that chondromas might be derived from intervertebral discs. Virchow (1857) first mentioned protrusion of a disc due to trauma, and in Ribbert's Pathology (1914) reference was made to small ecchondromas derived from intervertebral cartilages and found post mortem. However, special attention was attracted to these growths only after the surgery of the spinal cord had been developed, and the operation of laminectomy had become a surgical procedure without much danger. In 1928, Stookey published a report of 7 cases of "cervical ventral chondroma" which had been operated upon by the surgeons of the Neurological Institute. In all of the cases the growth was situated in the region of the fourth, fifth, or sixth cervical vertebra. Isolated case reports were published by Adson (1925), Pechy (1929), Veraguth (1929), Alajouanine and Petit Dutaillis (1929), Bucy (1930), and others.

In 1928, I made the suggestion that if the posterior borders of the intervertebral cartilages were carefully examined at autopsies, small prominences, not large enough to produce disturbances of spinal cord function, might often be found. In 1929, two papers based entirely upon autopsy material appeared, one by Andrae, the other by Schmorl.

Andrae examined the vertebral column at 368 autopsies, and discovered small prominences on the posterior borders of the intervertebral cartilages in 56 of the cases (15.2 per cent). The small nodules for which he used the term *Knorpelknötchen* were found most often in the bodies of females more than fifty years of age. As many as seven of these cartilaginous nodules were found in a single vertebral column. The swellings usually lay near the midline and sometimes laterally, they were always covered by the thinned out fibers of the posterior common ligament of the vertebrae, and were never large enough to have produced pressure upon the dural sac or the spinal cord within it. They were most often found on the posterior borders of the intervertebral cartilages of the middle and lower lumbar regions. He made no examinations of the intervertebral cartilages of the upper cervical spine.

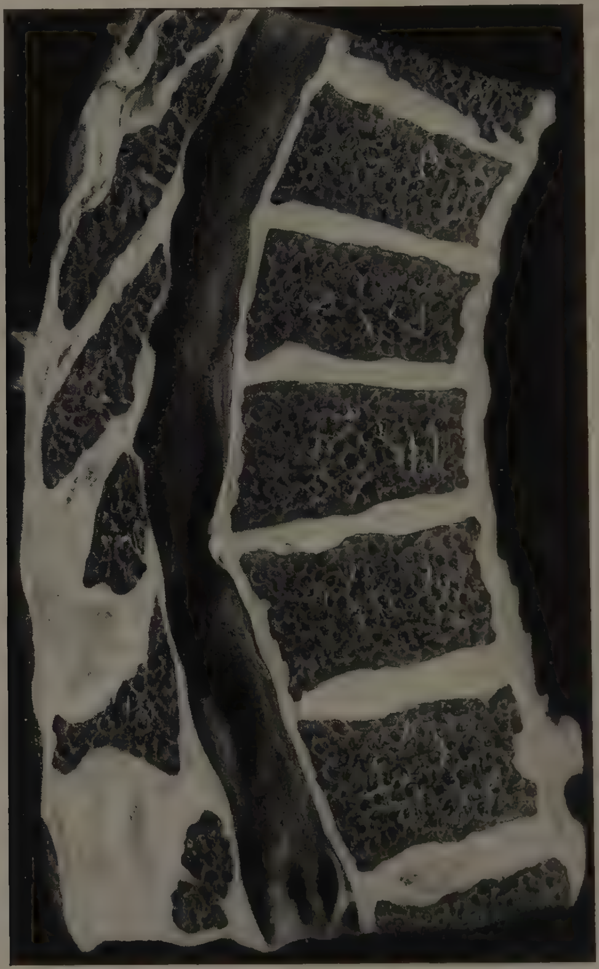

FIG. 227. An outgrowth from an intervertebral disc found at autopsy. (Schmorl.)

In his paper, Andrae did not describe the histology of the small growths in any detail. He declared that they occur secondary to destruction and tears of the annulus fibrosus, with prolapse of part of the pulpy nucleus into the vertebral canal and hyperplasia of the protruding cartilage. He was undecided whether trauma might have been an etiological factor in the process.

Schmorl agreed with the statements made by Andrae. He believed that the cartilaginous swellings are similar in nature to the small excrescences from the nucleus pulposus of the cartilages, which he had previously described as *Knorpelknötchen*, which often penetrate the surface plates of the cartilage and project into the bodies of the vertebrae. One of the illustrations in Schmorl's paper is reproduced in Figure 227.

Further investigations led to the acceptance of the views of Andrae and Schmorl: that in most instances the lesion which had been originally believed to be an ecchondrosis or a chondroma, was in reality a herniation of part of the nucleus pulposus, that this abnormality occurred very frequently although not exclusively in the lumbar region, and that prolonged backache and sciatica might be caused by this condition. In 1934, Mixter and Barr and Mixter and Ayer suggested that there was a relation between herniation of the nucleus pulposus in the lumbar region and sciatica or backache, and they showed that the diagnosis and situation of the lesion could be made by roentgenograms of the spine taken after the injection of lipiodol. This has been confirmed by a large number of investigators (Hawk, 1936; Barr, 1937; Love and Camp, 1937; Fincher and Walker, 1938; and others).

FREQUENCY, INCIDENCE, AND SYMPTOMATOLOGY

In our 275 cases of spinal cord tumor, there were 27 herniations of the nucleus pulposus* or 10 per cent. The average age of the patients was 50 years, the youngest 31 years and the oldest 68 years and about 80 per cent between 45 and 55 years of age.

Eighteen, or 66 per cent, were males, and 34 per cent were females. These figures agree with the figures given by Hawk, and are somewhat lower than the 77 per cent of males in 100 cases of Love and Walsh (1938).

In my experience, the herniation was in the lumbar region in 55 per cent, in the thoracic region in 18 per cent, and in the cervical in 27 per cent. In half of the patients, the lesion was between the third and fifth lumbar vertebrae, and occasionally above or below these vertebrae. Barr, Hampton and Mixter (1937) found the herniation between L 4 and L 5 in 60 per cent, and between L 5 and S 1 in 30 per cent of the cases. A history of a preceding injury to the back was obtained in less than one-third of the patients.

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* The early cases were classified as ventral chondromas.

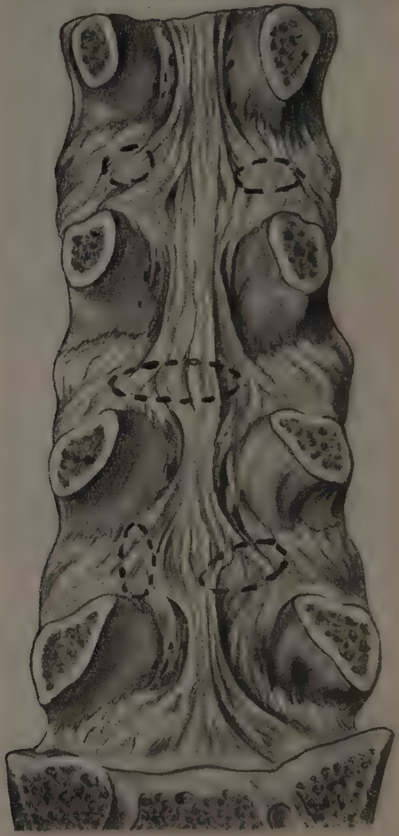

FIG. 228. The posterior longitudinal ligament of the vertebrae. The frequent situations of the ventral growths have been indicated by the circles inked on the original drawing. (Sobotta and McMurrich, Human Anatomy, 1906.)

Grossly, the lesion forms a prominence on the posterior surface of the intervertebral cartilage (Fig. 228) beneath the posterior longitudinal ligament, and is most often found lateral to the midline, where the ligament is weakest. The prominence may be fibrocartilaginous, or it may be partly calcified. It may be large enough to make pressure upon the spinal cord or caudal roots, or it may only press upon a single nerve root. With it are often associated secondary adhesive processes in the meninges, and almost regularly compensatory thickening of the corresponding ligamentum flavum (Fig. 229). Apparently there is no constant relation between the degree of hypertrophy of the ligamentum flavum and the size of the herniation of the nucleus pulposus. In advanced stages of cervical and thoracic lesions, there are secondary changes in the spinal cord.

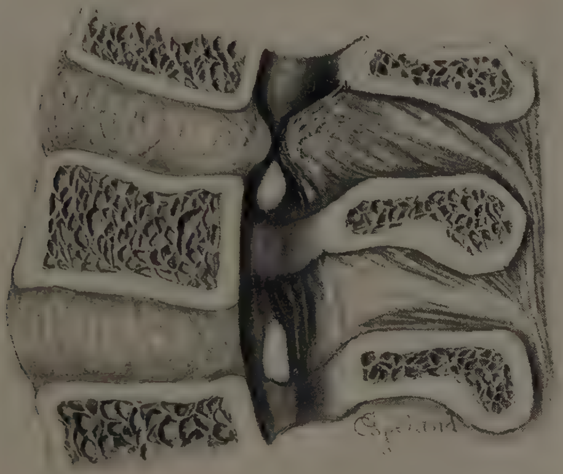

FIG. 229. Herniation of the nucleus pulposus with thickening of the ligamentum subflavum (diagrammatic).

The symptoms complained of depend upon the level of the lesion. The cervical herniations most often cause unilateral radicular pain or signs of compression of the spinal cord from in front. The patients with lumbar herniations complain of pain in the back or of sciatica, occurring in attacks or continuously for from one to ten years. The most usual complaint is pain in the lower part of the back extending down one or both limbs, followed by tenderness of the affected part of the spine, with perhaps some weakness or stiffness of the lower extremities. In some patients, pain in the back is the only complaint, and neurological examination is entirely negative. In most of the patients, however, there is more or less disturbance of power, reflexes, and sensation if they are carefully looked for, especially when the lesion is above the lowermost lumbar vertebrae. Tenderness along the course of the sciatic nerve is not unusual, and limitation of movement of the affected part of the spine due to muscle spasm is frequent.

When the lesion was lumbar, one Achilles jerk was diminished or lost in more than one-half of my patients, and one or both knee jerks diminished in about one-third of the patients. When the lesion was cervical, one biceps or triceps reflex was diminished or lost.

If carefully looked for, sensory disturbances are frequent. Slight diminution of cutaneous sensibility over the dorsum of the foot or of the calf, foot hyperesthesias or hypesthesias were not rare.

The information obtained from lumbar puncture was of real value as demonstrating a spinal lesion. Some degree of subarachnoid block was found in over 60 per cent of the patients, and some increase of total protein and globulin in the spinal fluid was found in more than one-half of the patients. Fincher and Walker (1938) found over 40 mg. of total protein per 100 cc. in more than 50 per cent of the cases, while Hawk found it in over 80 per cent. The highest total protein values in my series were between 100 and 400 mg., and values between 50 and 100 mg. were not rare. The globulin varied between 1+ and 4+. The spinal fluid was xanthochromic in only a few patients.

X-rays of the spine sometimes revealed definite changes; especially in the lumbar herniations. Narrowing of the affected intervertebral space (Chapter III) and loss of the normal lumbar lordosis were relatively frequent.

The diagnosis may often be suspected, but in the patients with herniation of lumbar cartilages with pain in the lower back or sciatica but without neurological disturbances, diagnosis and accurate localization can only be made by lipiodol studies.

The lipiodol examination for determination of posterior herniation of the nucleus pulposus, is made in the following manner:*

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* For this description, I am indebted to Dr. C. G. Dyke, of the Roentgen Department of the New York Neurological Institute. For full details see Chapter III.

"The entire examination is performed in the roentgen ray department. The patient is placed on the side with the head end of the table slightly elevated. Lumbar puncture is performed after the usual preparation of the patient, and 5 cc. of fluid is withdrawn from the spinal subarachnoid space. Five cubic centimeters of the heavy lipiodol is injected into the spinal subarachnoid space. After the completion of the injection, the patient is placed prone on the table. Fluoroscopy is then performed with the tube below the table and the screen above the patient. The lipiodol is usually found to lie opposite L 3 or L 4, and it forms a column about 8 cm. in length. The cephalic end of the patient is then slowly elevated so that the lipiodol will progress caudally. In the presence of a herniation of the nucleus pulposus into the canal, the lipiodol usually deviates to one or the other side of the canal and becomes pointed at the distal end. Further elevation of the cephalic end of the patient may cause the entire mass of the lipiodol to pass the point of obstruction which is usually opposite the intervertebral disc, but the filling defect in the opaque medium persists. In some patients a complete obstruction, or 90 per cent obstruction to the lipiodol occurs even with the patient in the erect posture. Lateral fluoroscopy in many instances shows a filling defect in the ventral margin, that is, the margin of the lipiodol in contact with the posterior longitudinal ligament. In order to study the canal above L 3 or L 4, the hips of the patient are elevated slowly, and the opaque medium is allowed to pass into the upper dorsal and even into the cervical area. It is emphasized that the patient be moved slowly, so that the column of lipiodol be not broken into a number of globules.

"Films are made whenever a filling defect or obstruction to the free flow of lipiodol is noted. These are made in the anteroposterior and lateral projections."

According to some investigators, the injection of air or oxygen into the spinal canal is a useful method for roentgenologic diagnosis, and the roentgen films will reveal some lesions quite satisfactorily. The advantage of using air or oxygen as a contract medium is that the gas is readily absorbed from the subarachnoid space; and if it can be demonstrated, especially in lumbar lesions, that as accurate a diagnosis can be made as by lipiodol, air or oxygen should be used instead of iodized oil.

It is sometimes difficult to distinguish these cartilaginous lesions in the cervical region from true spinal cord tumor. Usually, however, the symptoms and signs of cord or root compression are more clear cut in the latter. In some of our patients, an intramedullary expanding lesion was considered probable on account of muscle atrophies and dissociation of cutaneous sensibility.

Treatment

The only treatment is laminectomy and removal of the protruding cartilage by an extradural or transdural procedure. In the cervical and thoracic regions an extradural procedure is satisfactory only when the growth is laterally placed and easily exposed without much traction on the dural sac. Otherwise a transdural procedure is advisable. In the lumbar region, the cartilaginous mass can usually (but not always) be removed extradurally (Fig. 230).

As far as operations upon nucleus pulposus herniations in the cervical region are concerned, the following may be said:

In planning the surgical approach to a growth on the ventral aspect of the dural sac near the midline, the surgeon should fully appreciate the dangers to which the spinal cord will be exposed even if the necessary manipulations are carried out with care and skill. An extradural exploration for the exposure of the growth is more difficult than one done from within the dura after the sac has been opened. Although the growth often lies a little to one side or the other of the midline, it can rarely be satisfactorily exposed extradurally unless both the dural sac and the spinal cord have been raised and drawn to the other side. For such a manipulation a part of the spinal cord must be sufficiently mobilized to permit it to be drawn to one side. Therefore the dura must first be incised, and this procedure followed by mobilization and raising of the cord (Fig. 231).

The latter manipulation is accomplished by division of one or more slips of the dentate ligament after they have been grasped with fine mosquito forceps. The forceps are then used gently to raise the cord and to draw it toward the other side (Fig. 232). Occasionally, in addition, one nerve root has to be sectioned.

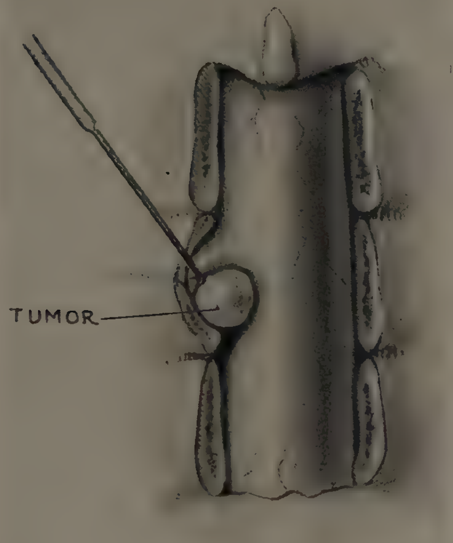

FIG. 230

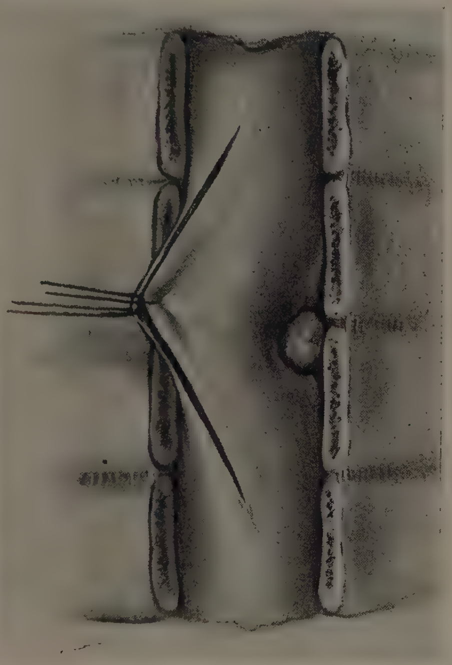

FIG. 231

FIG. 230. Exposure of a laterally placed ventral chondroma lying under a nerve root. (Elaborated from the surgeon's sketch.)

FIG. 231. A herniation of the nucleus pulposus a little lateral to the midline, exposed at operation. The dura was first incised and several slips of the dentate ligament divided before the growth was approached extradurally.

In order that all of this may be accomplished with the minimum of danger to the spinal cord, a wide opening into the vertebral canal must be made, and this can only be adequately accomplished by a bilateral laminectomy. Tumors on the anterior surface of the spinal cord can be, and often have been, successfully removed through a unilateral laminectomy, but I am convinced that at least as far as the extradural ventral chondromas are concerned, the removal of the laminae on only one side gives insufficient room for the necessary manipulations, does not allow as satisfactory a dislocation of the cord at the level at which the operation is being performed, and increases the risk of injury to the delicate cord tissue, unless the growth happens to lie well over to one side.

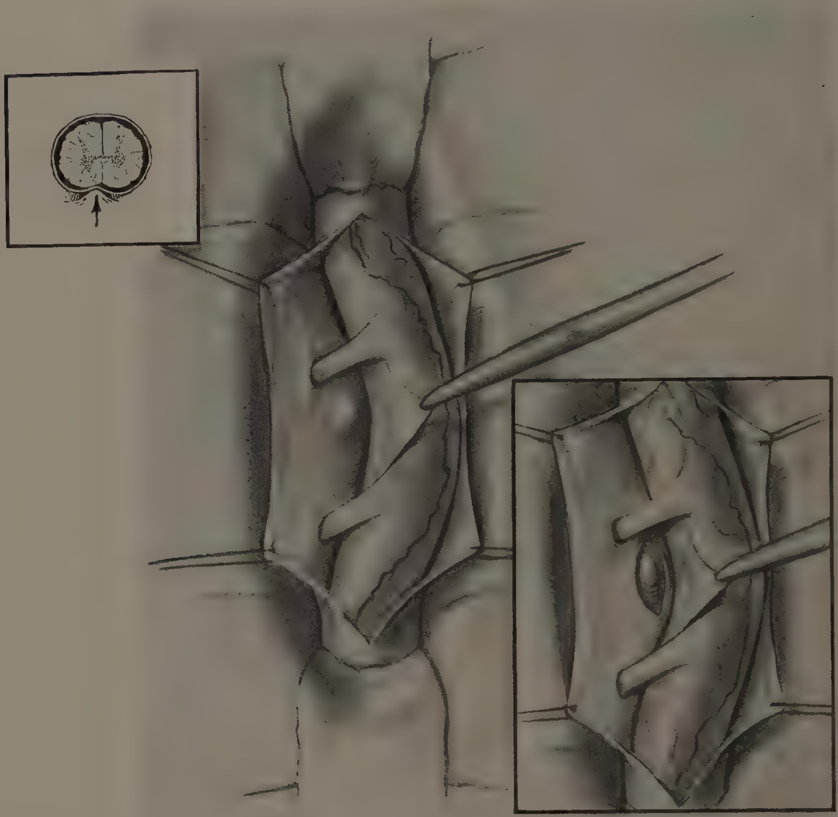

FIG. 232. Transdural exposure and removal of ventral chondroma. A. The prominence exposed by incision of the dura and a slip of the dentate ligament and lateral rotation of the cord. B. Incision of the posterior dura and exposure of the cartilaginous mass. Insert shows location of the mass. (Elaborated from the surgeon's sketch.)

It is likewise unwise, even if the dural sac has first been incised on its posterior surface and the slips of the dentate ligament have been divided, to attempt to raise and draw aside the dural sac and to expose the growth extradurally. There is much more venous bleeding from the posterior surfaces of the bodies of the vertebrae in this extradural approach and the sponging necessary to control the bleeding increases the risk of doing some harm to the cord within the dural sac.

As soon as the herniation has been exposed and the anterior dura incised, an incision is made in the posterior common ligament and the cartilaginous mass removed with a curette. It is often possible to enucleate the fibrocartilaginous mass in one piece. At other times a fragmentary removal is unavoidable. After the mass has been removed and bleeding, which is usually slight, has been controlled, the dural sac is allowed to fall back into place, and the posterior dural incision closed by suture.

Operations for herniation of a nucleus pulposus in the lumbar region can be accomplished, in most instances, by the extradural route. Not rarely, when the herniation has been exactly localized, the cartilaginous nodules may be removed through a small laminectomy of one vertebral arch. In the lumbar region, it is always advisable to excise the corresponding ligamentum flavum, which is often thickened and may be a contributing factor in the compression of one or other caudal root. If the nerve root is found to be thickened or much altered by adhesions, its sensory portion should be divided. The removal of a nucleus pulposus herniation in the lumbar region is seldom difficult, and the danger of the operation is small.

In a certain number of instances, there is an abnormal mobility between two vertebral bodies. In these instances a fusion operation to immobilize the affected vertebrae may be of advantage (Mixter).

Results of Treatment

In many of our patients, the operation has been too recent to permit one to state definitely that the symptoms have been permanently relieved.

The immediate relief of pain was usually marked and about 50 per cent of the patients were entirely freed from the pain or sciatica. In about 44 per cent of our cases, there was only partial relief, some pain or other symptom persisting. In 6 per cent, the removal of the protruding nodule did not relieve the patient.

Of the patients of Barr, Hampton and Mixter, 68 per cent were reported as well, 25 per cent improved, and 4 per cent unrelieved.

It is still too early to arrive at a final conclusion regarding the results of operative intervention for this condition. Undoubtedly many patients are entirely relieved, and it may be that in others, the symptoms which led to the surgical intervention were not due to the herniated disc.

GUMMA OF THE SPINAL CORD

Syphilis of the spinal cord is of interest to the neurosurgeon because occasionally the differentiation between it and tumor of the spinal cord may be difficult. Every neurosurgeon has probably had the experience of exposing the spinal cord on account of symptoms and signs which were finally found to be due to some form of syphilis of the cord. Even at the operation it may be difficult to recognize the nature of the disease. Furthermore, it happens occasionally, that the signs of compression of the spinal cord are so marked that an exploratory and decompressive laminectomy has to be done even though a diagnosis of gumma appears highly probable. As shown by Horsley many years ago, irreparable harm may be done by a gumma during the time that the patient is receiving antisyphilitic therapy. It must not be forgotten that an individual may have both syphilis and a spinal cord tumor, and in several instances I have removed spinal cord tumors from syphilitic patients. Finally, in some instances, it is impossible to distinguish between an extramedullary gumma and a neoplasm.

Syphilitic disease of the spinal cord most often begins in the meninges and extends into the substance of the cord. When such a lesion is exposed at an operation, there is usually a fusiform enlargement of the spinal cord with fine adhesions between the membranes. The enlarged cord often fills up the dural sac and is unusually soft, so that care must be taken not to injure the cord during the incision of the dura.

In one patient, in my experience, the lesion was limited to the dura in the cervical region. In this patient there was an irregular thickening of the dura around the cord, a true hypertrophic cervical pachymeningitis.

Sometimes the lesions are limited almost entirely to the nerve roots with only slight involvement of the meninges. Cases of this kind have been described by Nonne, and I have seen several instances in which there were a number of small enlargements along the course of the affected nerve roots.

The syphilitic lesions described may, occasionally, have to be differentiated from localized spinal diseases in which relief can be obtained by surgery. In most of the patients, the diagnosis can be made from the history, and the positive reactions of spinal fluid and blood by complement fixation tests.

Syphilitic meningomyelitis may simulate an extramedullary or intramedullary neoplasm, because a progressive course beginning with root symptoms and followed by cord involvement with spinal subarachnoid block and increased protein in the spinal fluid is frequent. Of more interest to the neurosurgeon is gumma of the spinal cord.

Gumma of the spinal cord (and of the brain) is always a late development of syphilis and is only met with many years after the primary infection.

There may be one or several gummatous tumors, and although these usually have their origin in the meninges, they may be primary within the substance of the cord. They may be met with at any spinal level, but are most often cervical or dorsolumbar. There are no clinical manifestations that are characteristic of compression of the spinal cord by gumma. Root pains due to irritation of posterior spinal roots with localized paralyses of lower motor neuron type followed by progressively increasing signs of cord compression dominate the clinical picture. If the gumma involves the roots of the cauda equina, the disturbances are those characteristic of a cauda equina lesion (*q.v.*).

Aside from the Wassermann reaction, a useful method in cases in which a syphilitic lesion is suspected is a trial period of three to four weeks of antisyphilitic treatment. If there is no definite improvement, exploratory laminectomy is indicated, or if the spinal cord symptoms advance rapidly in spite of antisyphilitic therapy, operative intervention should not be delayed.

CHAPTER XIII

PRIMARY AND SECONDARY DISEASES OF THE VERTEBRAE (INCLUDING VERTEBRAL TUMORS) WHICH MAY PRODUCE SPINAL CORD DISTURBANCES

THERE ARE A NUMBER OF PATHOLOGICAL CONDITIONS OF THE VERTEBRAE IN WHICH THE VERTEBRAL CANAL MAY BE INVADED BY the disease and the functions of the spinal cord disturbed either by direct pressure or by interference with the circulation of the cord.

Aside from traumatic lesions of the bones of the spine (Chapter IV) and osteomyelitis of the vertebrae (see extradural spinal abscess), the affections which are to be considered include arthritis of the spine, Kümmel's disease, Paget's disease, and tuberculosis of the spine.

ARTHRITIS OF THE SPINE

A variety of names have been given to chronic inflammatory processes which affect the vertebral column—arthritis deformans, spondylose rhizomélique, spondylitis deformans, osteo-arthritis deformans, and so on. In these diseases there are changes in the bodies and arches of the vertebrae and in the articulations. The edges of the bodies may become irregular, and the bodies of several vertebrae may become united by new bone. The affection may involve only a few vertebrae or the greater part of the spinal column. The main symptoms in all of these conditions are pain and stiffness of the back; cord symptoms are relatively infrequent.

The new-formed bone may cause a narrowing of the intervertebral foramina, compression of nerve roots and root pain. Localized areas of cutaneous hyperesthesia and localized muscle atrophies are not rare.

Symptoms due to interference with the functions of the spinal cord—hyperactive tendon reflexes and slight disturbances of cutaneous sensibility—are occasionally encountered, and when these disturbances are associated with a slight degree of subarachnoid block and slight increase of total protein in the spinal fluid, the possibility of compression of the spinal cord by a neoplasm has to be excluded.

Arthritic changes in the vertebrae are found in many individuals who have passed middle age, but in the majority of these cases, the arthritic changes do not produce any disturbances of the spinal cord or nerve roots.

In some individuals, however, there may be bony thickenings which project into and deform the vertebral canal and bony spurs may make pressure upon the dura and the spinal cord, or upon nerve roots. In individuals with marked vertebral arthritis, the deformation of the vertebral canal may produce a slight interference with the normal flow of cerebrospinal fluid in the subarachnoid space, sufficient to cause a slight increase in the total protein in the fluid. In these individuals a total protein of 60 to 70 mg. per cent is not unusual.

Spondylose rhizomélique is the name given by Pierre Marie to a disease which occurs almost exclusively in male adults and whose main characteristic is progressive anchylosis of the vertebrae. The disease usually begins with pain in the lumbar part of the spine and in the lower extremities. After a few months this part of the back becomes stiff, the pain disappears, and the patient is left with a more or less marked stiffness of the lumbar spine and of the hip joints. After the expiration of a few months or years, similar pain is felt in the cervical region and in the upper limbs, soon followed by stiffness of this part of the spine. The disease may begin in the cervical and later involve the lumbar vertebrae. The process then gradually extends over the thoracic vertebral column, and the patient is left with a stiff back, so that walking is interfered with and he is able to get about with great difficulty by the aid of canes and crutches.

The nature of the pathological process is unknown; it may follow gonorrhea or other infection. The lesion most often found is marked bone proliferation with calcification of the ligaments of the vertebrae.

Pain due to compression of the spinal roots may occur in the early stages of the disease. There is not, however, any tendency for narrowing of the vertebral canal to occur, and spinal cord symptoms are rare.

Kümmel's disease, or as it is often called, Kümmel-Verneuil disease, is characterized by atrophic changes in one or several vertebrae, which may occur after trauma to the spine. Months or years after a slight or severe injury of the back, the patient suffers from pain in the location of the former injury. Physical examination and roentgen films show that there is a gibbus due to atrophy of the body of one or of several vertebrae with angulation of the vertebral canal. The angulation of the canal may be followed by root pains or by more or less marked cord symptoms.

Paget's disease (osteitis deformans). In 9 cases of this disease we have observed marked changes in the vertebrae which caused the symptoms and signs of compression of the spinal cord, so that the question of a decompressive laminectomy was seriously discussed.

Of the 9 cases, 2 occurred in women and 7 in men. The average age was 58 years, and the average duration of the illness before the patient was seen was about one year. In all of the patients, there was weakness or paralysis of both lower limbs with sensory levels in the thoracic region.

The clinical course and the neurological examination do not show any definite differences between compression of the spinal cord in Paget's disease and compression from other causes. The diagnosis has to rest entirely upon roentgenological findings of the characteristic bony changes in the vertebrae and of similar changes in skull, pelvis, or other bones. Sometimes it is difficult to distinguish the roentgen appearance from that of metastatic carcinoma.

In the majority of instances, the symptoms and signs of spinal cord compression advance gradually, but occasionally there is a sudden onset of paraplegia due to the sudden collapse of the body of a diseased vertebra.

If the changes in the vertebrae are not too far advanced, the signs of cord compression may be greatly relieved by decompressive laminectomy. In one of our patients paraplegia and sensory disturbances disappeared entirely after a decompressive operation, and the patient remained well for more than ten years. Another patient has been entirely relieved for over four years. Other surgeons have had similar results.

If the examination gives satisfactory evidence that the cord symptoms are due to compression of the spinal cord by deformed bone, at a definite level, laminectomy is justifiable. The surgeon must always remember that the affected vertebra or vertebrae are already weakened by the pathological process and the indication and contraindication to operative intervention must be carefully weighed.

THE SPINAL CORD COMPLICATIONS OF TUBERCULOSIS OF THE SPINE

Tuberculosis may occur as a primary disease in the arches of the vertebrae, in the spinal dura mater, or in the substance of the cord, but its usual origin is in the bodies of the vertebrae. Compression of the spinal cord and the nerve roots may be due to the sinking together of the bodies of one or more vertebrae with angulation of the cord, or to projecting fragments of bone, extradural collections of pus, granulation tissue, sequestra or secondary pachymeningitis. As has been mentioned in another chapter, a localized serous meningitis may complicate tubercular bone disease.

Chronic edema of the cord over a localized area frequently accompanies tubercular disease of the vertebrae and dura, and is often followed by toxic softening of the cord at the affected level and by secondary ascending and descending degenerations in the fiber tracts.

Sometimes there are marked cord symptoms without any demonstrable macroscopic cord lesion, and a number of cases of this character have been reported in the literature. I have seen one patient with complete paraplegia in whom the postmortem examination failed to show any evidence that the cord had been compressed or diseased.

The symptoms may appear at any time of life from childhood to old age. Over 40 per cent of the cases occur in children before the fifth year, about 70 per cent before the tenth year, and over 90 per cent before the twenty-fourth year of life.

The disease usually begins in the bodies, but it may originate in the arches of the vertebrae and cause early cord symptoms. The progress of the affection is generally a slow one. The patients first complain of fatigue on slight exertion, or of pain in the back or in the distribution of one of the nerve roots. If the upper cervical vertebrae are affected, the first sign may be rigidity or stiffness of the neck or, in children, a retropharyngeal abscess. Some patients first consult the physician on account of a swelling in the back to one or other side of the spinous processes, while the occurrence of a girdle sensation or of root pains leads others to seek medical advice.

Tenderness of the spinous processes is very frequent. Unless the progress of the disease is interrupted by the treatment, a kyphosis with marked angular deformity occurs in more than one-half of the patients, followed by cold abscesses (retropharyngeal, retroperitoneal, psoas, etc.). Under appropriate treatment all of the symptoms may disappear and complete recovery occur. No patient can be considered cured, however, until two to four years have elapsed. Deformities of the spine remain in many instances.

If the disease involves the nerve structures within the spinal canal, the first symptoms are caused by root irritation or compression, although root and cord symptoms may appear at the same time. The distribution of the cord symptoms will depend upon the level of the cord lesion, and at different levels the symptoms will vary. When the uppermost cervical vertebrae are affected (malum suboccipitale), there may be symptoms referable to cranial nerves, with respiratory and cardiac difficulties, and all the signs of a high cervical cord lesion.

If the disease affects the lower cervical vertebrae, there may be all the symptoms and signs characteristic of that part of the spinal cord. In the lower dorsal region, the most frequent location of tubercular disease of the spine, the signs of a transverse lesion of the cord—spastic paraplegia of the lower limbs, anesthesia up to the affected segment, and disturbances of the vesical and rectal functions—are of frequent occurrence.

1. *Root pains* may exist for years as the only symptoms of Pott's disease. The roots are pressed upon either by bony deposits around the intervertebral foramina, by abscesses or masses of tubercular granulations, or by sequestra. The root pain may begin very suddenly with the sudden collapse of a vertebral body and the appearance of kyphosis, or insidiously, due to gradually increasing pressure upon the affected nerve roots. It is not rare for the symptoms to appear very suddenly after a trauma to the back. In one of my patients root pain appeared suddenly after the lifting of a heavy weight; and in another, pain in the abdomen appeared a few hours after a fall upon the back. Pain is rarely absent in Pott's disease, and is generally referred to the peripheral distribution of the nerve roots. In the cervical region occipital neuralgias are frequent; in the dorsal region intercostal nerve symptoms are complained of. Abdominal pain, as a result of root pressure in the lower dorsal and upper lumbar regions, has often been the cause of a mistake in diagnosis. A patient was once referred to my service, with fever and acute pain in the lower part of the right side of the abdomen, as a case of acute appendicitis. Careful examination showed that the pain in the right side was a skin and muscle tenderness due to posterior spinal root irritation, and the x-ray confirmed the diagnosis of tubercular disease of the lower dorsal vertebrae.

Another patient had for years a girdle sensation around the middle of the trunk as the only symptom of caries of the dorsal vertebrae. In still other patients a root hyperesthesia around one-half of the body or around the entire body, for a long time may be the only symptom of vertebral disease. Intercostal neuralgia is rarely a primary affection, but is usually secondary to intrathoracic or spinal disease. If a patient complains of intercostal pain, and no signs of an intrathoracic disease can be discovered, a careful examination of the spine by x-rays and a thorough neurological examination must be made. If an individual who has been losing flesh and who has slight evening temperatures develops an intercostal neuralgia without any other evidences of disease, the physician must be very suspicious that he has to deal with the early symptoms of tuberculosis of the vertebrae.

As recovery occurs by the substitution of new-formed bone for the tuberculous tissue, this bone may encroach upon the intervertebral foramina and cause root symptoms. In several instances I have seen patients who suffered from severe root pain due to this cause.

With marked kyphosis, the nerve roots may be stretched or pressed upon and severe root pains may result.

2. *The spinal cord* is involved in the disease, according to different authors, in anywhere from 3 to 18 per cent of the patients. The cord symptoms are due to a variety of causes. Actual tubercular disease of the cord is rare, although a few cases of solitary or conglomerate tubercle of the cord have been described, and I have once removed a tuberculoma from the substance of the lower dorsal cord. Tubercular abscesses and masses of granulation tissue on the outer surface of the dura are frequently met with. There is often a thick mass of granulation tissue adherent to the outer surface of the dura. Not so rarely the dura itself is the seat of a tubercular pachymeningitis which results in marked thickening, so that the cord is markedly compressed, with symptoms much like those of spinal tumor. The spinal cord symptoms may, on the other hand, be due to compression of the cord through the dura by dislocated bone, encapsulated masses of cheesy material, or new-formed bone which causes a marked narrowing of the spinal canal. However, there may be a great deformity of the spinal canal without any compression of the cord, and many patients in whom the disease has healed are left with a marked gibbus but without any spinal cord symptoms.

Ordinarily the cord symptoms appear very gradually. The patients begin to tire easily, there is a slowly increasing weakness and spasticity of the lower extremities with exaggeration of the muscle and tendon reflexes, girdle sensation, and root pains. The weakness in the limbs increases and well-marked signs of interference with pyramidal tracts are soon observed. A double Babinski reflex may appear very early and may persist for a long time before ankle clonus can be obtained. Root pains may be as severe in Pott's disease as in metastatic malignant disease of the vertebrae, so that the patient holds the affected portion of the spine rigid. The pain is increased by movement, by coughing, sneezing, or other jars to the spine. Hence stiffness of the neck in cervical Pott's disease, and rigidity of the back in dorsal or lumbar caries, is often a protective phenomenon. Aside from root pains and root hyperesthesias, sensory symptoms may not be observed for many years. If, however, the collapse of several vertebrae causes marked deformity of the spinal canal, the spinal cord may be compressed to such a degree that all of the symptoms and signs of a complete transverse cord lesion—complete motor and sensory loss, disappearance of all reflexes, paralysis of bladder and rectum—may appear, followed by trophic ulcers and decubitus. In the upper cervical region occipital neuralgia, anesthesia or hyperesthesia in the areas of distribution of the upper cervical nerves, bulbar disturbances (respiratory and cardiac difficulties), and paralysis of the upper extremities with rapidly advancing muscular atrophies are frequent. There may be paraplegia of all four extremities with sudden respiratory failure and death.

In the lower cervical, the dorsal, and the lumbar regions the characteristic symptoms of lesions of the spinal cord at the different levels are met with.

The compression of the cord is often incomplete and the motor disturbances are often much more prominent that the sensory disturbances. The Brown-Séquard symptom complex rarely occurs, although cases of this kind have been reported by Oppenheim and others. Transverse lesions of the cord most often occur in the advanced stages of tubercular disease, and the death of the patient is not long delayed.

Diagnosis

It is outside the scope of this volume to consider all of the symptoms of Pott's disease which are referable to the process in the bony structures—the fever and loss of flesh, the presence of tubercular lesions in other parts of the body, the formation of paravertebral abscesses, of psoas abscesses behind the peritoneum or in the groin, and so on. The x-ray examination will often show marked changes in the vertebral column, but in some cases the x-ray picture will give no evidence of the vetebral disease. The symptoms referable to the spinal cord and nerve roots are often so similar to those of other spinal diseases that the differential diagnosis may be difficult.

Malignant disease of the spine may present symptoms almost identical with those of Pott's disease. The stiffness of the back, the tenderness of one or more spinous processes, the root pains and the cord symptoms may be alike in both diseases. Tubercular affections of the lungs, bones, joints, and so on, and irregular fever occur rather in tuberculosis, although irregular rises in temperature are not so rarely observed in metastatic malignant disease of the vertebrae. Very severe root pains may occur in either affection, but they are more frequent in metastatic malignant disease. The history of previous malignant disease in other parts of the body is of value, but metastatic malignant disease is met with where the location of the primary new growth cannot be determined before death.

Many patients with spinal symptoms are treated for Pott's disease, and I know of several cases of vertebral or intradural tumor in which the spine had been immobilized in a plaster cast in the belief that the patient had a tuberculous disease of the vertebrae. The patients with spinal tumors are rarely improved by immobilization; in several cases that I have later operated upon, the symptoms were distinctly aggravated.

The differentiation between tubercular disease and osteo-arthritis deformans and other spondylitic changes of the spine, is sometimes not easy, but the x-ray picture will often make the diagnosis possible.

It may be difficult to differentiate the x-ray pictures of arthritis deformans from those of old tubercular disease of the vertebrae in which there has been an extensive new bone formation. From the therapeutic standpoint, however, the differential diagnosis is not important. When cord and nerve roots are markedly compressed by the new-formed bone, a decompressive operation is indicated, no matter what the nature of the disease.

In several instances the differential diagnosis between Pott's disease, aneurism of the aorta with bone absorption, and syringomyelia has presented some difficulties.

TREATMENT

The surgical treatment of the spinal complications of Pott's disease is called for only after conservative methods have had a long and thorough trial. Hygienic treatment, a high amount of nourishment, sufficient fresh air, and the like, are more important than drugs. The problem of local treatment is to hold the spine at rest, and this is accomplished by various orthopedic measures, such as rest in bed, plaster-of-paris jackets, extension, braces, and so on. The abscesses are treated conservatively by aspiration and injections of iodoform emulsions. Various methods have been described, whose object is to cause a bony union between a number of the spinous processes, and thus to permanently immobilize the affected part of the spine (Hibbs, Albee, Chipault, Cabot, and others).

The attempt to expose and to treat directly disease of the bodies of the vertebrae has given a few good results.

The removal of the arches of several vertebrae for the direct local treatment of the disease in the vertebral bodies should never be advised, because the already weakened spinal column may be thereby deprived of its last support. The exposure of the bodies of the vertebrae by costotransversectomy described by Menard, for the removal of foci of disease and the drainage of cavities in the bodies of the vertebrae, is a difficult and seldom justifiable operation. The bone disease cannot be eradicated by surgical means, for if the disease is extensive it is impossible to remove the entire bodies of one or more vertebrae.

The only hope for cure of the bone disease in the bodies of the vertebrae is by hygienic and orthopedic means, aided in special cases by operative immobilization of the spinous processes and laminae. As far as I can see, our only hope for a real treatment of the bone disease will be the discovery of a specific treatment for tuberculosis.

The early cord and root symptoms demand careful immobilization of the spine, and operative intervention is demanded only in those patients in whom the disease is at a standstill or is improving, if they develop increasing signs of compression of the cord. Root symptoms are often entirely relieved by repeated aspiration of a lumbar abscess with injections of 10 per cent iodoform emulsion. The abscess should not be incised unless there are already evidences of secondary infection with pyogenic organisms. It is almost impossible to prevent secondary infection of a tubercular abscess if an incision into it is made.

If, in spite of immobilization of the spine, the symptoms of compression of the cord have grown rapidly worse, and if the cord compression is not due to dislocation or collapse of the bodies of the diseased vertebrae, the opening of the spinal canal by the removal of the spinous processes and laminae should be done without delay. The laminectomy is done in the usual manner, but no more spines and laminae than are absolutely necessary should be taken away.

As soon as the laminectomy has been performed, the operator may find one of several conditions. If a collection of fluid is found on the side or in front of the dural sac, it must be opened by a small incision, the tubercular pus evacuated, and the cavity filled with iodoform emulsion which is injected by means of a syringe. The opening may then be closed by suture, or the edges of the incision may be sewed to the fascia of the back so that the cavity can be drained and be treated regularly after the laminectomy wound has healed.

Sequestra are sometimes found in the spinal canal and must be removed; most frequently they lie on the anterolateral aspect of the dura. The unopened dural sac must therefore always be drawn to one side, so that the anterolateral wall of the spinal canal can be examined. If the palpating finger discovers a sharp projection of bone on the posterior surface of one of the vertebral bodies, this prominence must be removed with rongeurs. For this purpose the dural sac may have to be lifted up from the body of one or several vertebrae, and it is occasionally necessary to divide one nerve root before this can be satisfactorily accomplished. The nerve root must be cut as far away from the dura as possible, so that no opening into the dural sac is made.

There may be very lively oozing from the venous plexus between the anterior surface of the dural sac and the posterior surface of the body of the vertebra, but this is usually easily controlled. After the prominence of bone has been removed, the dural sac is allowed to slip back into place, and the laminectomy wound is closed in the usual manner.

The dura should never be incised, if tubercular disease has been found outside of it in the spinal canal. If, however, there is no evidence of disease on the outside of the dura to explain the cord symptoms, then a small exploratory incision is made and the arachnoid and cord examined. Should a localized serous meningitis (adhesive arachnitis) be found, the fluid is evacuated and the dural sac again closed.

The results of operation for the spinal cord complication of Pott's disease have, in the main, been very unsatisfactory. Decompressive laminectomy, in a few patients, has prevented an irremediable transverse lesion of the spinal cord from pressure of a pachymeningitis or from cold abscesses or sequestra in the spinal canal. In most of the patients, however, the benefit derived from the operation has been a questionable one. Unless there are special indications, a laminectomy for the spinal symptoms of tubercular spondylitis should be done only when all other methods have failed to afford relief.

There are some patients who, years after the tubercular process has healed, develop signs of gradually increasing compression of the cord. The x-ray picture generally shows that the spinal canal has been narrowed by a new growth of bone. In these patients a decompressive laminectomy with the removal of the new-formed bone, wherever it presses upon the dural sac, will be followed by great improvement, but operative intervention should not be too long delayed.

PRIMARY TUMORS OF THE VERTEBRAE

Steinke (1918) collected 330 cases of compression of the spinal cord by new growths; of these 45, or 13.6 per cent were primary tumors of the vertebrae. This percentage is probably much too large.

In the material of Schlesinger (1898), there were only 107 cases of vertebral tumor among 35,000 autopsies; while Antoni (1936) found in 6840 postmortem reports by Henschen only 12 cases of primary tumor of the vertebrae (1 case of chondroma and 11 cases of cavernous angioma of the vertebrae). In my personal experience, based mainly upon clinical and operative material, primary tumors of the bones of the spine were unusual, angioma and chondroma were the most frequent, and osteoma and giant cell sarcoma were rare.

Primary *chondroma* which produces special cord disturbances may occur in young adults. The diagnosis is easily made from roentgen films of the spine. The spinal disturbances may occur acutely during the course of a few weeks, or the interference with spinal cord function is slowly progressive due to gradually increasing pressure upon the spinal cord.

What has just been written concerning chondromas is equally applicable to the osteomas. *Osteoma* of the vertebrae which produces spinal cord symptoms is quite unusual, and up to 1936 about 15 cases had been reported. The diagnosis is usually made from roentgen films of the spine, and treatment must consist of laminectomy and partial or complete removal of the osteoma.

In both chondroma and osteoma of the vertebrae, the result of surgery depends upon the stage in which the patient is operated on, and the completeness with which the tumor can be removed. If the chondroma or osteoma is derived from the vertebral arches, complete removal is often possible, but even if the removal is incomplete, there may be absolute relief, which may last for many years. If the growth is derived from the posterior surface of the body of a vertebra, the exposure and removal of the neoplasm may require a degree of elevation of the dura which might endanger the spinal cord within the dural sac. In these cases, the laminae on the side from which the osteoma or chondroma is to be approached, must be removed almost to the articular processes in order to allow of the manipulations required for the removal of the growth. Sometimes it is advisable to open the dura and to divide one or several slips of the dentate ligament to permit of elevation of the cord into full view.

Hemangioma of the vertebrae was considered to be rare until the characteristic x-ray appearances of these growths in bone were described (Hitzrot, 1917; Perman, 1926). Recent investigations (Makrycostas, 1927; Töpper, 1928; Bucy and Capp, 1930) have shown that vertebral hemangiomas that have produced no symptoms during life are often found at autopsy. However, marked symptoms of spinal cord compression may occur, as in the cases described by Muthmann (1903), Bailey and Bucy (1929), Junghanns (1932), and others. I have personally seen and operated upon three young adults with vertebral angioma which had produced symptoms of spinal cord compression.

The lesion may affect either a single vertebra or a number of vertebrae. It is usually situated in the vertebral body, but may extend into the transverse processes and the arches.

The disease has been found most often in young adults and the two sexes are about equally often affected. If, as is often the case, the angioma does not cause any neurological disturbances for many years, the patient may be seen at a more advanced age.

For several years the symptoms may be limited to pain in the back or pain radicular in distribution. If cord symptoms appear, they have the character of slowly advancing compression, distinguishable from extradural or intradural tumor only by the characteristic changes found in roentgenograms of the affected part of the spine.

As it is not rare for more than one vertebra to be affected, the radicular pain may involve at least several root areas, so that the pain is more diffuse than that ordinarily observed in extramedullary cord tumors.

When the disease extends from the body of a vertebra into the vertebral canal, the symptoms produced are those of compression. The compression of the spinal cord is due to the extension of the process extradurally, to a secondary and distinct extradural hemangioma (as in one of my patients), or to the partial collapse of a vertebra. The motor, sensory, and reflex changes are those usually found in compression of the spinal cord, and spinal subarachnoid block with characteristic changes in the fluid withdrawn by lumbar puncture is not unusual. More or less manometric block was observed in each of the three patients I have operated upon.

The characteristic changes seen on the roentgen films are described in Chapter III. It may be stated here that the changes consist of a reduction in the density of the bone between parallel vertical trabeculae which are increased in density (Bailey and Bucy).

If the characteristic x-ray changes are found in an individual who has no signs of compression of the spinal cord, the indications are for thorough roentgen therapy. By this means, pain or radicular disturbances may be to a great extent or entirely relieved.

If there are signs of increasing spinal cord compression, operative intervention may be indicated. The surgeon, however, must fully appreciate the difficulties that may be encountered from the marked vascularity of the bone, and must be prepared for the control of bleeding. The laminae are sometimes so soft that bone wax is not as efficacious as usual. Free use must be made of high frequency current cauterization, and, if the bleeding is excessive, the operation may have to be done in several stages.

A number of cases have been reported in which surgical intervention, followed by roentgen therapy, has given satisfactory results—complete or marked relief of symptoms lasting for a number of years (cases of Perman, 1926; Bailey and Bucy, 1929; Junghanns, 1932; Hammes, 1933; and others).

Chordoma, a tumor derived from the remnants of the notochord, may occur in any part of the vertebral column, but has been found most often in the upper cervical vertebrae (as an extension from a growth in the occipital bone) or in the sacrococcygeal region.

Notochordal rests are found in the nuclei pulposi of the intervertebral discs, and the vertebral chordoma is probably derived from these remnants of the notochord. The tumor is a malignant one which extends into the soft tissues around the affected bone and may grow into the vertebral canal and produce spinal cord compression with all of its symptoms. The growth usually affects more than one vertebra and ordinarily can only be incompletely removed. Removal of the growth, even if only partial, may cause improvement in symptoms, but recurrence is almost the rule. It is still uncertain whether radiotherapy has any definite influence upon the growth.

Primary giant cell sarcoma is a form of tumor which is rarely encountered in the vertebral column. Up to 1934, Antoni (1936) collected 36 cases from the literature, of which 9 were cervical, 13 thoracic, and 14 were lumbosacral. I have operated upon 2 patients with this type of growth. In both instances the tumor involved the spinous process and laminae of a cervical vertebra and formed a prominent mass on the back of the neck; it caused root pain but no spinal cord disturbances.

It is well known that this type of tumor is usually benign, and it is probable that the implication of the term sarcoma is not correct. The growths are not genuinely malignant and have no tendency to recur after their removal. The roentgen appearance is characteristic (Chapter III), quite similar to that of the osteitis fibrosa of von Recklinghausen.

These growths usually begin in the vertebral arches but they may originate in or involve the bodies of one or more vertebrae, and secondarily invade the vertebral canal. When this is the case, there are spinal cord symptoms due to compression.

If the lesion involves the spinous process of a vertebra, complete extirpation is possible. This was the case in my two patients, both of whom recovered and have remained well for many years. On the other hand, involvement of the body practically excluded surgical removal. In these patients, roentgenotherapy may be tried; it has been beneficial in several reported cases.

Myeloma is a rare form of malignant tumor of the bone marrow, which may affect one or more vertebrae. The disease usually occurs in adults at or past middle age and may produce spinal cord symptoms. When there are multiple tumors in the flat and short bones—ribs, sternum, scapulae, skull, and vertebrae—the diagnosis is easily made. The multiple bone involvement, the presence of Bence-Jones bodies in the urine, and progressive anemia and cachexia make a highly characteristic clinical picture. However, there are cases in which for a considerable period of time, the growth is confined to the vertebral and extradural tissues, and makes itself known by signs of compression of the spinal cord. The vertebral form of the disease is not rare; during the past decade, cases have been reported among others by Wakely (1931), Jacox and Kahn (1933—2 cases), Klemme (1933—5 cases); Busser and Lichtenberger (1933), Ellerman and Schroeder (1933), Denker and Brock (1933—3 cases).

The typical picture is that of an individual past middle age, who develops rapidly advancing signs of compression of the spinal cord in the thoracic region. The examination shows a more or less marked spinal subarachnoid block with characteristic alterations in the cerebrospinal fluid withdrawn by lumbar puncture. Roentgenograms show erosion or partial destruction of the bodies of one or more vertebrae, similar to that seen in metastatic malignant disease. If there is no other clinical or roentgenological evidence of a primary disease, the possibility of myeloma has to be considered. The presence of Bence-Jones bodies in the urine make the diagnosis certain, but albumoses may not be found on repeated examinations.

Unless there are multiple bone lesions demonstrated by x-ray examination, laminectomy is generally indicated. At the operation, in addition to evidences of bone lesions, there is usually a large extradural tumor which may surround the dural sac on all sides. The growth is usually grayish red in color, of fairly firm consistency and elastic to the feel. The growth is not adherent to the dura, may involve the arches of the vertebrae, and cannot be completely removed.

The operation is usually followed by great improvement, which may last for a number of years. I have operated upon three patients: one succumbed in less than one year with multiple tumors and pathological fractures; the second patient lived for two years; the third has remained well for over three years. All three patients received thorough and repeated roentgen therapy after the operation.

While improvement lasting for a number of years has occurred in cases of myeloma limited to one or several vertebrae, the prognosis is poor in most of the patients. The growth recurs locally, either in some other part of the spine or in the flat and short bones of the entire body. The average duration of life in multiple myeloma is about two years.

SECONDARY AND METASTATIC TUMORS OF THE VERTEBRAE

While secondary growth in the bony spine may occur in sarcoma, hypernephroma, echinococcus disease, and so on, the most frequent and important cause is metastatic carcinoma. Metastases in the vertebrae occur especially often in carcinoma of the breast, prostate, and thyroid. The secondary growths after cancer of the breast may occur in any part of the spine; metastases from carcinoma of the prostate are most frequent in the lower thoracic or lumbosacral vertebrae.

The secondary growths are usually primarily situated in or around the body of one or several vertebrae and may extend into the vertebral canal and make pressure upon the spinal cord within the dural sac. More rarely, the dura itself is invaded by the growth or there is a tumor nodule within the substance of the spinal cord.

The growth at first affects the spinal nerve roots, and it may not involve the spinal cord for a long period. The spinal cord disturbances are sometimes due to direct pressure upon the cord through the dural sac, but more often they are caused by interference with the blood supply of the spinal cord or perhaps by a toxic effect upon the nervous tissue.

Some authors state that the spinal cord disturbances are most often due to gradual compression, but others (most recently Antoni, 1936) believe that more sudden involvement of the spinal cord is most often observed. My experience would support the latter view. In the most recent statistics of Antoni (1936) which comprise 229 cases of metastatic carcinoma of the spine, 72, or 31 per cent, were secondary to primary disease of the female genitalia (breast, uterus, ovaries); and 56, or 24 per cent, were secondary to primary disease of the prostate.

In the majority of instances, the first clinical symptom is pain, located in the back or in one of the large joints or having the character of a radicular neuralgia. It is often very severe. There are cases, however, in which disturbances of the spinal cord appear without preceding radicular pain. In every patient with radicular pain of unknown origin, the possibility of spinal metastasis from a growth in some other part of the body must be kept in mind, and the breast and uterus, thyroid and prostate must be carefully examined. It need hardly be mentioned that such pain in a patient who has been operated upon for a tumor of the breast or the prostate, no matter how long before, must lead to suspicion of spinal metastasis.

Pain may be the only symptom for months or even for years, and the patient may succumb to other metastases (liver, lung, etc.) without ever having shown any symptoms of spinal cord involvement.

When spinal cord symptoms appear, they occur in one of two ways: (1) Most often, there are the signs of a rapidly advancing spinal cord lesion which may cause complete flaccid paraplegia within one or a few days. At first, the paralysis may be complete with no disturbance or only slight disturbance of cutaneous sensibility, excepting perhaps radicular hyperalgesia or diminution or loss of sensation. Soon, however, sensory disturbances occur, followed by the signs of complete transverse lesion of the spinal cord. In these patients the acute symptoms may be due to the collapse of a diseased vertebra, which causes sudden and marked pressure upon the dural sac and spinal cord, easily demonstrable by x-ray examination and by manometric tests which show a complete spinal subarachnoid block. Often, however, the acute onset and rapid progression are due to an indirect effect of the metastatic growth upon the spinal cord (interference with vascular supply?). Under these circumstances, the fluid pathways in the subarachnoid space are not obstructed.

2. In a smaller number of patients, a long period of rheumatoid or radicular pain in the back or in one or the other limb is followed by increasing signs of interference with spinal cord function. These cases may be difficult to distinguish from those with progressive pressure upon the dural sac and spinal cord due to a primary extradural neoplasm. Manometric tests will demonstrate more or less interfer-

ence with the patency of the spinal subarachnoid space; roentgen films may not show any evidence of bony abnormality.

In proved metastatic carcinoma of the vertebrae, there is very rarely, if ever, any justification for the attempted removal of the malignant disease. Most of the cases in which the attempt was made were patients in whom the correct diagnosis was made only at the operating table.

Treatment can only be palliative, mainly directed to the relief of pain. This may be accomplished by roentgen therapy, paravertebral injections of alcohol, or chordotomy. The operation of chordotomy (*q.v.*) consists of division of the anterolateral tracts on one or both sides. The indications and contraindications to chordotomy are discussed in Chapter XVI. The operation should be performed only on patients whose condition is such that it is probable that they will live for a number of months or longer.

In rare instances, metastases in the vertebrae and vertebral canal have been found, in which the histological structure of the growth was that of normal *thyroid* tissue. Some writers believe that these growths are derived from misplaced thyroid rests, while others support the view that they are true metastases. There is a question also, whether some of the recorded cases were metastases from a carcinoma of the thyroid gland in which the histology of the spinal growth is similar to that of normal thyroid tissue. There is nothing especially characteristic in the clinical course of these thyroid growths of the vertebrae. At the operation, performed on account of the signs of cord compression, an extradural growth derived from the bone is found, which histologically is reported as normal thyroid tissue. Many of the patients have received roentgen therapy after the operation, and a large number have been completely relieved of symptoms by the partial or complete removal of the extradural growth followed by x-ray therapy.

Another unusual secondary lesion of the vertebrae which may cause symptoms of a lesion of the spinal cord, is *aneurism of the aorta*. In a recent review of the subject, Shemkin (1938) found 46 case reports of aortic aneurism which had eroded one or more vertebrae and produced disturbances of the spinal cord. I have seen one patient with a complete paraplegia from this cause.

In almost 25 per cent of the recorded cases of aneurism of the aorta, there was more or less vertebral erosion but there were spinal cord disturbances in less than 1 per cent of these.

The patients were usually males of middle age and a history of syphilitic infection was frequent. In the majority of instances, the aneurism was in the descending thoracic aorta, and the characteristic symptoms of substernal or scapular pain with cardiac disturbances preceded the occurrence of spinal cord symptoms by one or by several years.

The evidence of spinal cord involvement occurred either gradually or suddenly. In the latter instances, the sudden onset frequently followed a slight trauma to the back and was caused by either collapse of an eroded vertebra or by acute epidural hemorrhage. The symptoms are usually those of a more or less advanced spastic paraplegia which may end in a flaccid stage.

If a patient with known aortic aneurism develops spinal cord symptoms, the diagnosis is clear. The diagnosis can always be made by x-ray and by fluoroscopy.

The outcome is usually fatal within a few months after the occurrence of spinal symptoms, but cases have been recorded in which life was preserved for many months.

Antisyphilitic therapy has no influence upon the progress of symptoms, and surgical intervention is always contraindicated.

CHAPTER XIV

PRIMARY ABNORMALITIES OF THE SPINAL BLOOD VESSELS

PRIMARY ABNORMALITIES OF THE SPINAL BLOOD VESSELS MAY BE DIVIDED INTO (1) ANEURISMS OF THE SPINAL ARTERIES; (2) aneurismal varices of the arteries and veins; (3) varicosities of the spinal veins. With any of these lesions, the vessels within the substance of the cord may also be affected.

ANEURISM OF THE SPINAL ARTERIES

This is a rare condition, of which only a few cases have been reported (Heboldt, 1885; Guizzetti and Cordero, 1908; Balo, 1925; Sargent, 1925; and others). In Heboldt's case, a girl of fifteen developed meningitis following erysipelas. The autopsy disclosed several small aneurisms in addition to diffuse multiple abscesses of the brain. The patient of Guizzetti and Cordero had an aneurism of the anterior spinal artery between the first and second dorsal vertebrae. The aneurism was about 2 cm. long and had ruptured. Balo's patient was an adult man with spinal cord disturbances, in whom a diagnosis of tabes had been made. The patient died of pneumonia and at the autopsy an aneurism derived from the posterior spinal artery was found. There was subarachnoid hemorrhage.

Sargent's case was that of a male of 44 years, who gave a history of two years of progressive spinal cord symptoms with a level in the cervical region (Fig. 233). Laminectomy was attempted but was abandoned because of excessive hemorrhage. The muscles and bones contained numerous dilated tortuous thin-walled arteries and the condition was that of a diffuse aneurismal varix. The patient died shortly after the operation. The autopsy disclosed that the enlarged vessels extended into the spinal canal and that there was an aneurism containing recent blood clot which compressed the spinal cord at the level of the seventh cervical segment.

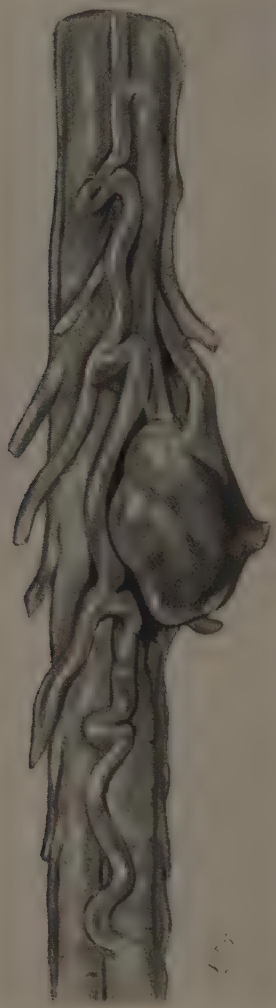

FIG. 233. Aneurism at the level of the seventh cervical segment (Sargent).

In addition to the cases of aneurism of the spinal vessels, a number of cases of aneurism of one of the vertebral arteries which caused the signs of compression of the spinal cord have been described. The following case, from my records, is an example:

Aneurism of the right vertebral artery that caused signs of extramedullary compression of the medulla and upper cervical cord. Laminectomy under local anesthesia. Death after five days from respiratoy paralysis.

June 21, 122 (M.H. No. 45034). Male, an electrician, 54 years of age, admitted May 30, 1922.

Duration and first symptom. One and one-half years before admission, the patient began to complain of pain in the back of the neck.

Progress of symptoms. A few months later, he began to suffer from numbness and paresthesias in the fingers of both hands, followed by increasing loss of sensation in the hands. Soon afterward he felt a straplike constriction around the neck followed by an increasing loss of power and sensation in both upper extremities. Six months before admission to the hospital, the lower limbs began to feel numb and he gradually lost power in the limbs, so that, three months later he became bedridden. All four extremities became stiff and the loss of strength and feeling became more and more marked. He soon became absolutely helpless and had to be fed, as he was unable to use his arms. A few months later he began to have difficulty in breathing, and at about this time lost control of his bladder. He became rapidly worse and had some difficulty in swallowing during the last few weeks previous to admission.

Physical examination. An emaciated man who lies in bed perfectly helpless. The pupils are equal and react to light and accommodation; there is no nystagmus; there is slight pallor of both optic discs. There is distinct hypesthesia of both corneae and distinct weakness of the masseter, temporal, and internal pterygoid muscles.

Motor and reflex disturbances. The patient cannot turn his head to the left as well as he can to the right. He cannot approximate his chin to his chest. There is a marked spasm of the scaleni muscles on the right side.

Both upper limbs are very spastic, the left more than the right. Slight flexion and extension are possible at the left elbow, and he can slightly flex and extend the second and third fingers of the left hand. There is very slight flexor power at the right elbow and of all of the fingers of the right hand. Pectoral jerks +++, right greater than left; deltoid +++; biceps, triceps and wrist jerks +++. The power of the lower limbs is better than that of the upper limbs. He can lift the left leg two feet from the bed, and the right leg somewhat less. He can abduct the left thigh, but not the right. Active flexion and extension at the knees and ankles is very weak but preserved. He can move the toes of both feet. Both limbs are very spastic. The abdominal reflexes are lost, but the cremasterics are preserved. Double patellar, knee and ankle clonus, doubt Babinski and Chaddock.

Cutaneous sensory disturbances. These are very extensive and involve the entire body from the second cervical dermatome down. Articular sense is lost in the right lower limb and much diminished in the left; preserved in both upper extremities excepting in the hands. Vibratory sensibility is lost below the level of the sixth rib and diminished up to the clavicles; it is lost in the right upper extremity and is much diminished in the left.

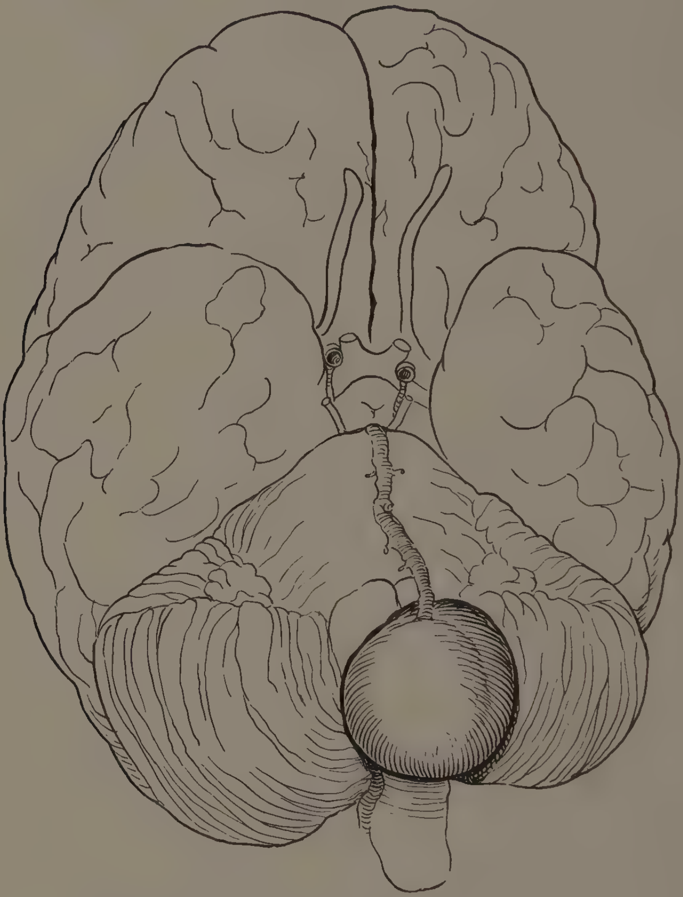

FIG. 234. Aneurism of the right vertebral artery.

Laboratory tests. Roentgenogram negative. Wassermann in blood and cerebrospinal fluid negative. Fluid obtained by lumbar puncture contains no cells; globulin is not increased.

Clinical diagnosis. Neoplasm within the foramen magnum and extending down to the fourth servical segment, making pressure from before backward (Dr. Abrahamson).

June 24, 1922. *Laminectomy* (C 1, 2, 3 and 4) under local anesthesia. Incision of dura. There was no tumor on the posterior surface of the cord, but an obstruction to the probe was met with above, just within the margin of the foramen magnum. On account of the poor condition of the patient, further exploration was discontinued.

Subsequent notes. The patient's condition remained good for two days. He then developed aphonia and bulbar symptoms and died from respiratory paralysis five days after the operation.

At the postmortem examination a large aneurism of the right vertebral artery was found. The aneurism measured 3 x 4 cm. and was partly filled by organized blood clot. The lower part of the medulla and the first, second and third cervical segments were markedly flattened (Fig. 234).

The symptoms produced by aneurism of the spinal vessels which cause compression of the spinal cord cannot be distinguished from the symptoms produced by extramedullary tumors. In none of the cases in which surgery was attempted was the nature of the lesion disclosed at the operation. If the aneurism is small, it may not produce symptoms, and the first appearance of spinal cord disturbances may be acute, due to subarachnoid bleeding or hemorrhage into the substance of the cord.

ANEURISMAL VARICES OF THE ARTERIES AND VEINS OF THE SPINAL CORD

There has been considerable confusion between the cases that may be called aneurismal varices of both arteries and veins, and those in which the pathology is limited to the veins. From a study of the cases reported in the literature under various names—telangiectasis, angioma, hemangioma, varicocele of the spinal cord, angioma racemosum—it is possible to divide the cases into two groups: arteriovenous aneurismal dilatations of the spinal vessels which are rare, and varicosities of the spinal veins which are frequently encountered.

Abnormalities of the spinal vessels in which both arteries and veins were dilated and tortuous were found in the cases of Brasch (1900), Raymond and Cestan (1902), and Elsberg (1925). In the cases of Lorenz (1901), Meyer and Kohler (1917), Benda (1922), Guillain and Alajouanine (1925), it was impossible to be certain whether arteries and veins or only the veins were involved in the process.

The patient of Brasch was an adult of 59 years who developed paralysis of the lower limbs and sensory loss with incontinence of urine during the course of two years. The patient died soon after admission to the hospital. At the autopsy there were found dilated and tortuous arteries and veins on both the anterior and posterior surfaces of the cord from the first thoracic to the lumbar vertebrae. Some of the changed vessels extended into the spinal cord.

In the case reported by Raymond and Cestan, a man 30 years of age had a slowly progressing weakness of the limbs with symptoms indicating a lesion of the cervical cord. The patient died before a laminectomy could be performed. The postmortem examination showed that the cervical cord was compressed by a mass of dilated and tortuous arteries and veins. The abnormal vessels extended into the substance of the cervical cord.

The following case, observed and operated on by me, belongs in this category:

A boy 13 years of age was well up to the age of 11, when an accidental fall resulted in what was called a "concussion" of the spine, which confined the patient to bed for several days. One year later he began to suffer from pain in the left thigh and soon afterward from pain in the right leg. The lower limbs gradually became weak. Loss of control of the bladder and bowels developed.

Examination revealed that the lower extremities were spastic, the knee jerks hyperactive, bilateral ankle clonus and Babinski. The abdominal and cremasteric reflexes could not be elicited. Cutaneous sensibility was diminished over the lumbar and upper sacral dermatomes.

A laminectomy at the level of the eighth, ninth, tenth and eleventh thoracic vertebrae revealed an intradural mass of tortuous blood vessels (Fig. 236C). The mass was 4 cm. long. A vein and artery were observed to be entering the mass from above, and two large vessels could be traced from the lower end to the cauda equina. Some of the vessels entered the substance of the spinal cord. A part of the mass of blood vessels was excised but there was no improvement in the symptoms and signs.

The symptoms produced by arteriovenous varices are indistinguishable from those produced by other extramedullary or intramedullary lesions. When the vessels within the cord substance are markedly involved, the clinical picture may be that of an intramedullary lesion. If the mass of vessels is entirely extramedullary, the symptoms may be very like those of a localized extramedullary lesion. Carefully made manometric tests should show some interference with the normal flow of fluid in the spinal subarachnoid space. An injection of lipiodol might outline some of the vessels. In the case of the boy in whom the lesion was exposed at an operation there was a history that the spinal symptoms were more marked when he was in the upright position than when he was recumbent, and this difference may have been significant.

In these arteriovenous varices, as far as is known, nothing has been accomplished by surgery. In the patient upon whom I operated, the ligation of several of the vessels did not alter the symptoms. Perhaps, in the future, roentgenotherapy will be found to be of benefit.

VARICOSITIES OF THE SPINAL VEINS

Dilatation and more or less localized varicosity of the spinal veins is frequently the result of obstruction, at a higher level, to the return flow of blood in the spinal veins. This is often observed below the level of extramedullary cord tumors, but it may also be the result of venous obstruction from a swollen spinal cord, a narrowed vertebral canal, or an adhesive process of the membranes. This type of dilatation of the spinal veins is secondary, and the venous congestion will disappear soon after the obstruction has been removed.

Varices of the spinal cord not secondary to obstruction of the spinal veins at a higher cord level, occur with considerable frequency. The recognition of the lesion is important from the surgical standpoint because spinal varices have often been found as the only discoverable abnormality at operations performed for suspected tumor of the spinal cord.

Up to the year 1912, a few cases had been described: one by

Gaupp (1888), which he called "hemorrhoids of the spinal pia mater," in which the lumbar cord was compressed by dilated and tortuous vessels; one by Jumentié and Levy-Valensi (1911) in which, at the postmortem examination of a patient with paraplegia, numerous dilated and tortuous veins were found over the cervical and upper thoracic cord. Krause (1911) exposed a mass of dilated veins during the course of a laminectomy in a paraplegic individual, and Lindemann (1912) described a case of varices of the veins of the spinal pia mater and cord as the cause of a total transverse lesion.

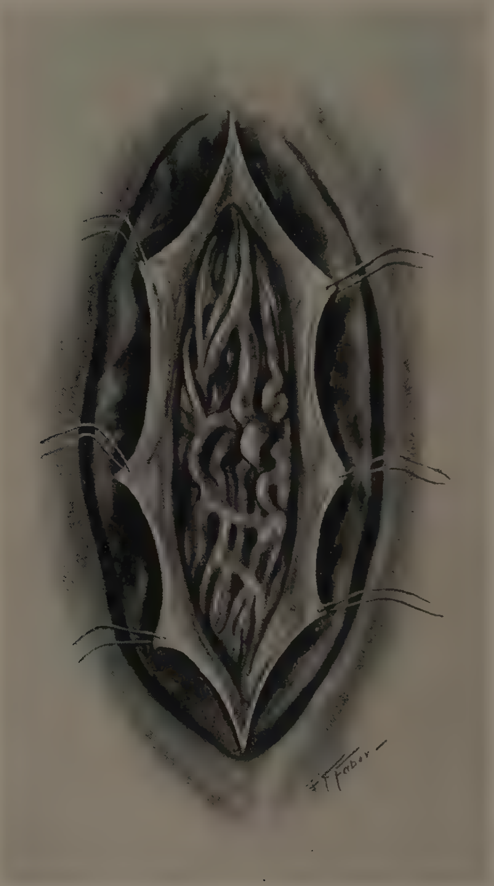

Fig. 235. Varicose veins of the spinal cord. The authors describe the case as one of telangiectasis of the spinal cord. (Spiller and Frazier, *Archives of Neurology and Psychiatry,* 1923.)

In 1915, Cobb described and illustrated a case under the title "hemangioma of the spinal cord"; and in 1916, I described a series of 6 cases in which at the operation, enlarged and varicose spinal veins were found. Since 1916 a large number of papers on spinal varices have been published (Spiller and Frazier, 1923, Fig. 235; Lapora-Gonzalo, 1924; Sachs, 1925; Globus and Doshay, 1929; Ritter, 1931; Schminke, 1936; Puusepp, 1938; and others).

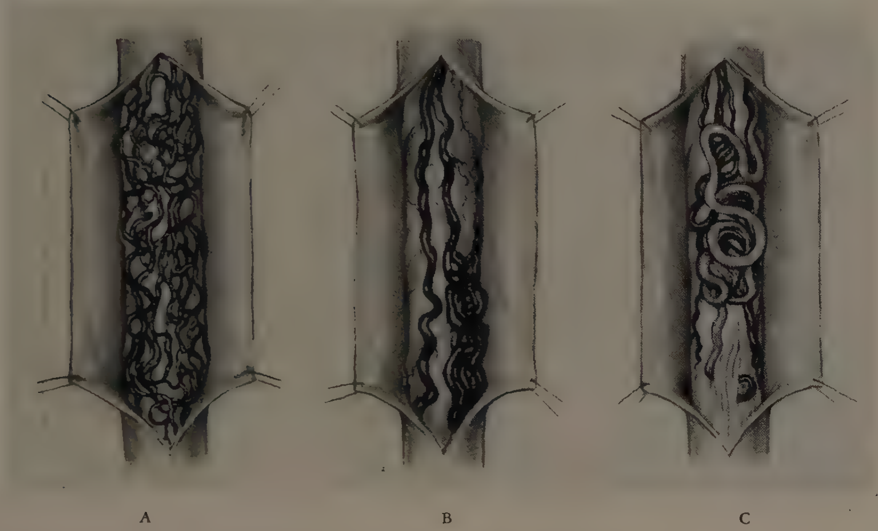
A B C

FIG. 236. A and B. Varicosities of the spinal veins. C. Atteliovenous varix of the spinal vessels.

Various theories have been advanced as an explanation for the occurrence of spinal varices. Some writers have considered them a congenital anomaly, but against that theory is the fact that, with the exception of Cobb's case (a child of 8 years) all of the patients have been adults. Others have entered the realm of speculation when they wrote of the possible effect of trauma in individuals with a congenital predisposition to venous dilatations. The only argument to support the view that there may be a congenital factor is the fact that in two reported cases (Cobb, 1915; Alexander, 1922) there were associated cutaneous nevi.

Although isolated, enlarged, and perhaps tortuous veins may be met with on the outer surface of the dura secondary to extradural or intradural tumors, true spinal varices are intradural and usually subarachnoid. However, in a few instances, I have seen the mass of dilated veins on the outer surface of the arachnoid membrane. Not rarely, dilated vessels may be seen to enter the substance of the spinal cord.

The enlarged veins are usually found on the posterior surface of the cord. A single vein may be involved, or even the branch of a posterior spinal vein that accompanies a spinal root. More often both posterior spinal veins are enlarged and tortuous, so that there is a mass of dilated and varicose veins which overlies one or several segments of the cord (Fig. 236 A, B), or the changes in the veins extend over a large number of cord segments. If the varices are localized, they are most often found in the cervical and especially the lumbosacral levels. In rare instances, one of the veins is so greatly enlarged as to form a mass which compresses the cord.

Symptoms and Diagnosis

The lesion is generally encountered in males, although a few cases in women have been reported.

The development of symptoms is similar to and sometimes indistinguishable from that produced by spinal cord tumor, but in many of the patients, the history dates back so many years that tumor is improbable. Thus, in a number of our patients, the first symptoms had appeared from five to twelve years before—a period that is unusual for a spinal growth. When, on the other hand, the symptoms have existed for only one to two years, the history is very like that of a case of tumor of the spinal cord.

The first symptom is usually pain in the back or in one extremity, which has the character of root pain. It may be less when the individual is recumbent, and may be made worse by forced expiratory movements such as straining or coughing. The onset of pain is usually acute and is followed by disturbances in the power and sensation of one or both lower limbs. Improvement soon begins, and after a few weeks all of the disturbances may have disappeared. Then for a number of weeks, months or years, the individual may remain well. Perhaps the symptoms return again followed by a symptom-free period. Finally, the symptoms persist and become more aggravated with marked disturbances in the power of the limbs, in the reflexes and in sensation, and with diminution or loss of control of the functions of the bladder.

Some individuals are seen when their main complaint is pain that is radicular in character. Others, who have no pain, consult the neurologist because of motor and sensory disturbances in the limbs. I have seen two patients with spinal varices over the conus and roots of the cauda equina whose main complaint was loss of control of the functions of the bladder and bowels.

The objective disturbances found at examination vary with the location, extent, and degree of change of the varicose veins. If the veins that accompany one or several nerve roots are markedly enlarged but the veins on the surface of the cord are relatively small, very little may be found on neurological examination. If, on the other hand, spinal cord symptoms are due mainly to pressure upon the cord by a mass of distended veins or by one greatly distended vein, the clinical picture may be that of a localized spinal cord compression. If there are many enlarged intramedullary veins and marked intramedullary changes, the clinical picture may be that of an intramedullary disease.

There is nothing especially characteristic in the motor disturbances, but the sensory examination will often show several distinct levels of diminution or loss of sensation.

In the majority of instances, the roentgen examination fails to demonstrate any pathological changes in the bony spine. In a few of our patients, however, there was a distinct atrophy of the pedicles of one or of several vertebrae. This atrophy was found in vertebrae below the vertebral level of the sensory disturbances. I have gained the impression that this discrepancy between the upper vertebral level of disturbances of cutaneous sensibility and the vertebrae whose pedicles are atrophic, is especially frequent in spinal venous varices.

Lumbar puncture and manometric tests may show various degrees of spinal subarachnoid block, but the block is rarely complete, and if the enlargement of the veins is not marked, there may be no interference with the free flow of fluid in the subarachnoid space. The fluid obtained by lumbar puncture may be normal in all respects; but in at least 75 per cent of my patients, it was slightly yellow, and in 60 per cent the total protein was increased. Total protein values of 70 to 80 mg. per cent were frequent, and values of 100 to 200 were not unusual.

Some authors believe that lipiodoloscopy is of value for the diagnosis of varicose veins of the spinal cord. We have not been able to substantiate the claim that there is anything characteristic in the way in which droplets of lipiodol are held up in the lower thoracic or upper lumbar regions.

In our experience, a clinical diagnosis of spinal varices can rarely, if ever, be made with certainty, although quite often the condition may be suspected. A combination of a history dating back many years (with perhaps several intermissions in the symptoms), slight or no subarachnoid block, slightly xanthochromic fluid and moderate increase of total protein, with atrophy of the pedicles of one or several lumbar vertebrae below the vertebral level of the disturbances of sensibility, must make one think of the possibility of spinal varices. If the history of the illness is of one or two years' duration, and the symptoms and signs do not quite fit into the picture of spinal cord compression by tumor, the possibility of spinal varices must be kept in mind. Recently an optical instrument has been devised by Pool, which can be introduced into the subarachnoid space. With this instrument it has been possible to recognize tumors and other lesions, including varicose veins of the conus and cauda equina. The method appears promising, but the future will have to show whether myeloscopy will be reliable for the diagnosis of varices in the lower part of the spinal canal.

Treatment

In the majority of instances reported in the literature of the subject—and this has been my personal experience—spinal varices have been found in patients in whom tumor was suspected or in whom the laminectomy was an exploratory procedure. Not rarely, in the preoperative records, varicose veins of the spinal cord had been mentioned as a possibility. A few writers have claimed that they were able to make the diagnosis.

It is still an open question whether patients with advanced intramedullary symptoms of many years' duration, due to varicose veins of the cord, should ever be subjected to operation. If this diagnosis could be made with certainty, for example, by myeloscopy or by some other method, I believe that operative intervention would be contraindicated. It has been our experience that in these individuals there are many vessels which penetrate the substance of the cord. These vessels cannot be ligated without great danger of causing much additional intramedullary change with the symptoms and signs of a complete transverse lesion.

When varicosities of the spinal vessels are exposed at a laminectomy, the first question to decide is whether the enlargement and tortuosity is limited to the veins or whether both arteries and veins are affected. When the lesion is limited to the spinal veins, one must make certain that the changes are not due to an obstruction in the canal at a higher level. The appearance of the posterior spinal veins below the level of a cord tumor is quite different from that of spinal varices. In the former, the posterior spinal veins may be slightly enlarged and varicose, but never to the degree encountered in the latter condition. Even if the spinal varix involves only one posterior spinal vein, the vessel is usually larger and more tortuous than it is in venous obstruction.

In a relatively small number of cases, the varix is localized, so that normally appearing veins are seen above and below the affected area. I have encountered a localized mass of varicose veins most often over the conus and roots of the cauda equina. Usually the mass of varicose vessels extends for a considerable distance beyond the area exposed by the laminectomy.

In many instances, enlarged veins emerge from the substance of the cord; in others they run with the posterior nerve roots to the dura openings. If the enlargement is localized and the enlarged veins are few in number, and if there are not any vessels entering the cord tissue, it is allowable to ligate and excise one or several of the enlarged veins, or to apply silver clips to them. This must be done with care, for the walls of the veins are thin and easily torn, and control of the resulting bleeding may be troublesome. If the varix is at all extensive, it is better to do nothing with it. If root pains have been severe, it is sometimes of advantage to divide, or, better, to crush, one of several posterior spinal roots, taking care not to injure an enlarged vein if it accompanies the root.

Theoretically, roentgen therapy should have an effect upon the varicosities, and this form of therapy should always be tried.

In my experience and in that of most neurosurgeons, little has been accomplished by surgery. Several of my patients in whom a single locally enlarged and tortuous vein, exposed at the laminectomy, was excised, were relieved so completely that they were able to work and remained well for from three to twenty years. A few others were relieved of root pain by division or crushing of appropriate posterior spinal roots. The occasional result of a laminectomy in extensive spinal varices was an improvement in symptoms and signs which lasted for from one to four years; in these patients, nothing was done when the varices were exposed, and the result must have been due to the decompressive effect of the operation. Beneficial results such as these occurred only occasionally. In the large majority of the patients, there was no improvement after the operation.

CHAPTER XV

LAMINECTOMY

THE TECHNIQUE OF THE REMOVAL OF TUMORS OF THE SPINAL CORD AND OF OTHER SURGICAL LESIONS WITHIN THE VERTEBRAL CANAL have been described in other chapters. In this chapter, the methods to be used for the opening of the vertebral canal will be described. Following this, I shall discuss the indications for certain surgical procedures upon the spinal cord and nerve roots, and the technique of the operations.

HISTORICAL NOTES

The operation of laminectomy dates back to early times, and there is little doubt that injured spinous processes and laminae were removed in the very dawn of surgery.

Paul of Aegina, in the seventh century, recommended incision of the skin and removal of fractured laminae. In 1626, Fabricus Hildanus removed a sword blade from the lumbar vertebrae by an open operation. Ambroise Paré and many of his contemporaries operated for fracture of the spine, and Heister removed the arches of the vertebrae and exposed the spinal dura.

Chopart and Desault (1779) were among the first to speak of "trephining between the spinous and transverse processes," but the first typical laminectomy is said to have been performed in 1814 by Cline, who used the well-known Hey saw to cut through the laminae.

For many years, however, the operation of laminectomy was considered a very dangerous procedure. And even in the era of antisepsis and asepsis, this opinion has been held by many down to our own time. In 1823, Astley Cooper made the far-seeing statement: "Though I may not live long enough to see the operation frequently performed, I have no doubt that it will be occasionally performed with success." To this Charles Bell responded: "We must submit to hear many strange proposals for the improvement of our profession in the present day from young men ambitious of notice, but that a man of Sir Astley's years and station should talk as he has done before students, and give them his authority for laying a patient upon his belly, and by incisions laying bare the bones of the spine, breaking up these bones, and exposing the spinal marrow itself, exceeds all belief."

Operations for injury of the vertebral column soon became more and more frequent, and attempts were made to relieve the spinal symptoms of Pott's disease by "trephining of the spine."

Later on, operations on the spine were performed for new growths and echinococcus disease of the arches of the vertebrae, which had made pressure upon the spinal cord. The earlier operations could hardly be called by the name of laminectomy, because they consisted merely of the exposure and removal of injured or diseased bone. Gradually, however, surgical intervention consisted, more and more often, of the removal of spinous processes and laminae, in order to expose disease or injury underneath them.

When the knowledge of the localization of functions in the cord became sufficiently advanced, the stage was set for the development of the operation of laminectomy in tumors of the spinal cord, and the opening act was the justly celebrated operation of Victor Horsley in 1887. Horsley made the skin incision in the median line over the tips of the spinous processes. After the muscles had been separated from the spines and laminae, a primary bone opening was made with a trephine, and the spines and laminae partly rongeured away with forceps and partly removed with hammer and chisel.*

In 1889, Dawbarn proposed an osteoplastic procedure in which an H-shaped incision was made in the soft parts and the spines and laminae turned upward and downward with the soft tissues. Three years later Urban proposed turning a skin-muscle-bone flap upward after making a U-shaped incision in the soft parts.

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* The story of the early development of the technique of the operation of laminectomy is well told by Chipault in his "Études de chirurgie médullaire," published in 1894.

Marion, and soon afterwards Cavicchia, described methods in which a flap was made of the skin, muscles and spines and laminae, and turned to one side. Bickham, in 1905, gave an elaborate description of such an osteoplastic procedure.

Osteoplastic operations were also described by Mixter and Chase, Ollier, Borchardt, and others. In 1921, a method was described by means of which the spinous processes were included in a skin-muscle-bone flap, while the laminae were rongeured away in the usual manner. Aside from the fact that an osteoplastic operation is a more complicated and time-consuming procedure, every one of the osteoplastic methods described interferes with free access to the dura and the cord, and none of them has any real advantages.

Theoretically, of course, it is correct surgery to leave the parts operated upon in as nearly as possible a normal condition after the surgical intervention, but practically, in many instances, this cannot be done.

These osteoplastic procedures were devised because it was believed that the complete removal of the spinous processes and laminae in a laminectomy would leave behind a weakened vertebral column and a cord more exposed to injury. As the main support of the spinal column is due to the bodies of the vertebrae and the muscles and ligaments which surround them, the column is very little, if at all, weakened by the removal of three or four of the arches. Nor are the movements of the column interfered with if the muscles and fascia are carefully sutured together at the close of the operation. If the different muscle and fascia layers are carefully sewed together and if they heal by firm union, they form a perfect protection for the cord and dura.

With the same object of preserving intact as much of the vertebral column as possible, A. S. Taylor, Allessandri, and Bonomo, independently, described the operation of unilateral laminectomy. In this method, only one side of the spinous processes and laminae is exposed and the laminae of one side are rongeured away laterally to their junctions with the transverse processes, and medianward to the bases of the spinous processes (Fig. 237). This unilateral operation, or hemilaminectomy, gives a fair exposure of the cord and is of value when posterior spinal roots of one side are to be divided. It is obvious that in a hemilaminectomy the amount of space is limited as compared with that in a complete laminectomy; and I believe that this unilateral procedure is usually inadvisable in those operations in which the maximum of room is needed in order to reduce the handling of the spinal cord to the minimum.

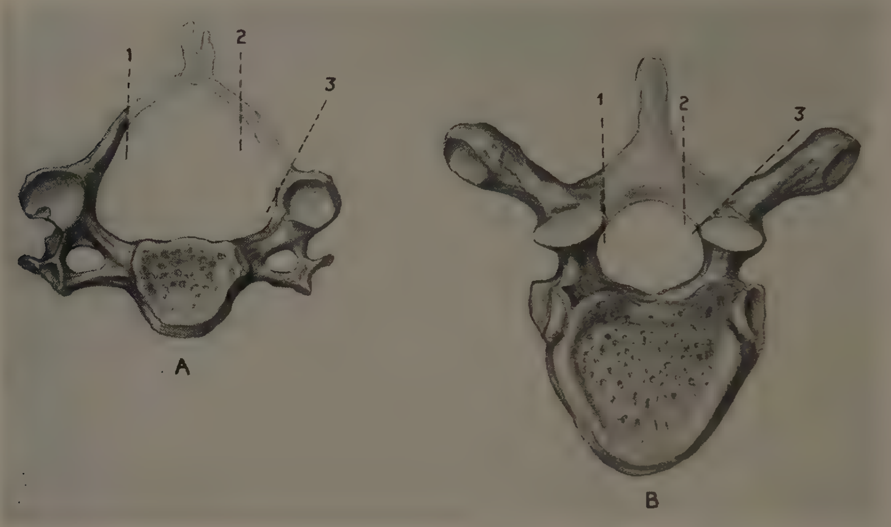

FIG. 237. A cervical (A) and a thoracic (B) vertebra. The amount of bone removed in an ordinary laminectomy is shown between broken lines *1* and *2*. Between the broken lines *1* and *3*, is shown the amount of bone to be removed for the approach to the anterior surface of the cord from the left (on the right in the diagram).

The operation of laminectomy consists of the exposure and removal of the spinous processes and laminae of one or more vertebrae.

PREPARATION OF PATIENT

The preparation of a patient for a laminectomy under anesthesia should be a simple one. It does not differ from that for any other surgical procedure. The back is shaved over a large area, and if the operation is to be done in the cervical region, the hair is shaved up to the level of the external occipital protuberance.

ANESTHESIA

For the ordinary laminectomy, avertin followed by ether given on an open mask is the preferable anesthetic. When the patient is in the prone position for a laminectomy in the cervical region, nasal intubation may be useful; and in high cervical operations, anesthesia by the intratracheal method is occasionally of value, especially in a patient with a short, heavy neck.

If a general anesthetic is contraindicated, the laminectomy can be done painlessly under local anesthesia. Frazier published an excellent method by which the spinal roots are anesthetized between the transverse processes of the vertebrae; and a very satisfactory anesthesia of the field of operation can be obtained by Frazier's method.

A fully adequate anesthesia can be obtained, however, by avertin anesthesia followed by the successive infiltration of each layer of tissue with novocain before it is divided. As soon as the skin and fascia have been incised and the muscles have been separated from the spinous processes and laminae, an injection of 1 per cent novocain between each two transverse processes on either side will make the removal of the spines and laminae painless. The incision of the dura is not painful. If much manipulation around posterior spinal roots is necessary, the roots may be blocked by applications or injections of ½ to 2 per cent novocain solution.

As in all operations, blood pressure, pulse, and respiration are continually controlled during the surgical procedure. It is a good plan to have a blackboard in the operating room, on which the nurse can chart her findings, so that the operator at a glance, can know the facts.

POSITION OF PATIENT

The position of the patient must be varied according to the part of the vertebral column in which the laminectomy is to be performed. In general, the patient must be placed in the prone position,

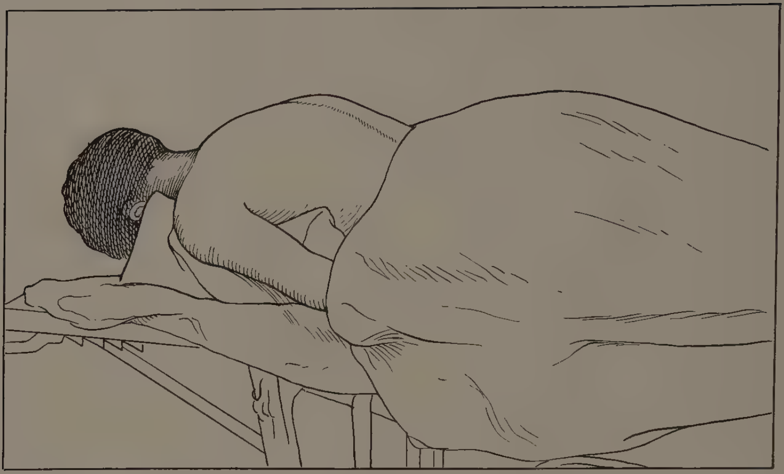

FIG. 238. The prone position for laminectomy.

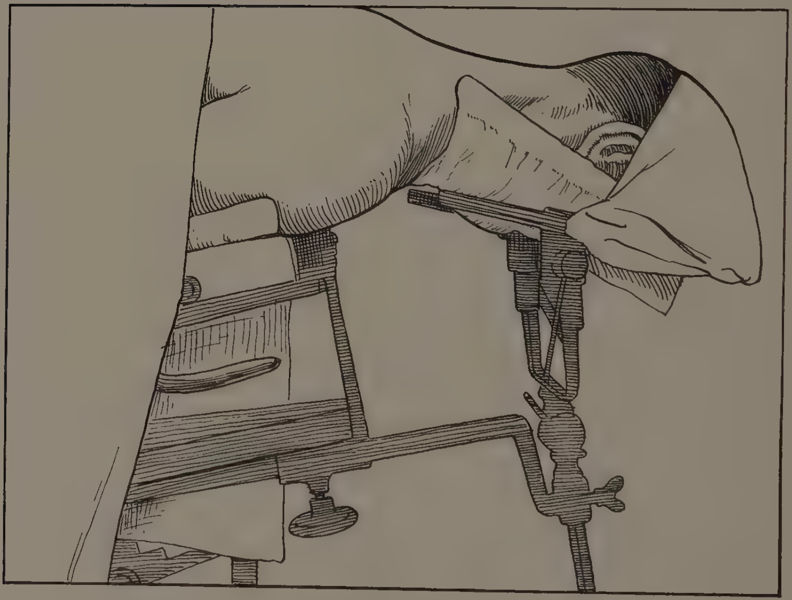

FIG. 239. The position of the patient for cervical laminectomy.

supported on pillows so that the chest remains free and respiratory movements are not interfered with. Hard round pillows are used to make more prominent the part of the spine in which the laminectomy is to be done (Fig. 238). This postural kyphosis makes the exposure of the laminae easier.

For laminectomy in the cervical and upper thoracic regions, the patient should be placed in the same position as if a suboccipital craniotomy were to be performed. The head is supported on an outrigger or a headrest (Fig. 239).

If the operation is to be performed in the middle or lower thoracic or the lumbar spine, the patient's back should be made prominent by a hard roller cushion placed under the abdomen.

When there are marked contractures of the lower limbs, considerable ingenuity may be required in order to have the patient in a favorable position for the operation. When the spastic contractures are extreme and the legs are acutely flexed upon the thighs, it may be necessary to place the patient more or less on one side instead of in the prone position.

SPINES AND LAMINAE TO BE REMOVED

The determination and locating of the spines and laminae to be removed is a matter of great importance. As is well known, the segments of the cord do not lie on the level of the correspondingly named vertebrae, and there is, at best, considerable variation in the relation between the two, as has been shown by Muskens, Reid, and others. For this reason it is always advisable to remove three spines and laminae. A serviceable "rough and ready" rule is that in the lower thoracic region, each segment of the cord lies three vertebrae higher, and in the upper half of the thoracic and in the lower cervical cord, two vertebrae higher, than the correspondingly named spinous process.

Although there are many individual variations, Figure 4 shows the relation between the spines of the vertebrae and the segments of the cord in the majority of instances. If the operator is in doubt as to the exact location of the segments of the cord he desires to expose, it is always advisable to go one spine higher than the spines that have been located.

In order to locate a spinous process, it is advisable to begin counting from an easily recognizable landmark. The spine of the seventh cervical vertebra (vertebra prominens) is easily felt, but occasionally the spine of T 1 is also prominent. If there are two prominent spines, the upper is that of the seventh cervical vertebra. From this spine, one must count downward until the middle one of the three desired spines has been located. In order to be positive that this is the desired spinous process, one begins counting upward from below, beginning at the spinous process of the fourth lumbar vertebra as a landmark, which, as is well known, is on a line with the highest points of the crests of the ilia. However, especially in stout individuals, errors may be made. Therefore we have made it a rule to have a roentgenogram taken before the operation; on this the vertebral level of the suspected disease is marked by a small lead arrow. For the roentgenogram the lead marker is attached to the skin by adhesive plaster, 2 or 3 cm. from the midline. At the same spot, a drop of a colored dye is injected into the skin. The roentgen film will then indicate the exact spine at the level of the arrow and the cutaneous dye injection.

TECHNIQUE OF OPERATION

The skin incision is made in the median line, directly over the spines to be removed, and is rapidly deepened until the tips of the spines have been exposed. The fascia and muscles are then cut off close to the spines, the cutting edge of the scalpel being directed against the sides of the spinous processes. The edges of the fascia and muscles are pulled away from the spines by sharp retractors, the latter being inserted deeper and deeper as more of the soft tissues are freed from the bone. By means of a flat broad raspatory or broad chisel the soft tissues with the periosteum covering the spines and laminae are cleanly scraped away from the bone until the laminae are well exposed. There is sometimes considerable bleeding during these manipulations, but it can be checked by temporarily packing gauze into the cavity or by the use of the electric current. Artery forceps are only rarely required.

The same procedure is now carried out on the other side. The gauze packings and the sharp retractors are then removed and spinal automatic retractors inserted (Fig. 240). When the blades of the retractors are opened, all bleeding is controlled.

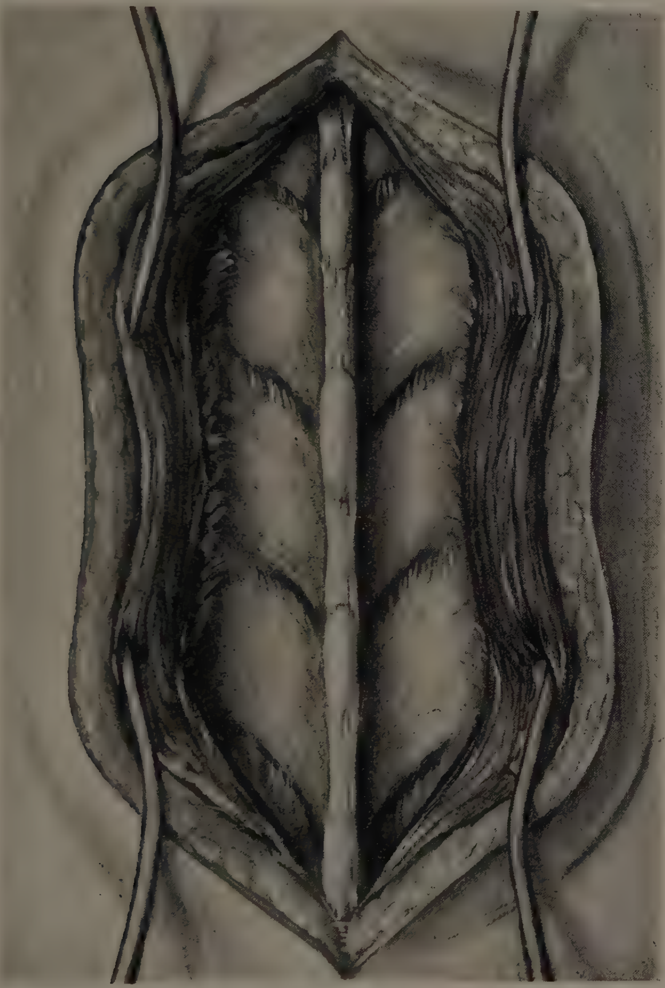

FIG. 240. Laminectomy. The incision in the soft parts has been made and the muscles separated from the spinous processes and laminae. The automatic retractors in place expose the laminae and control bleeding from the muscles.

The interspinous ligaments are now divided with a scalpel, and the spines, beginning with the lowest one, bitten off at their bases with giant rongeur forceps (Fig. 241). The laminae are then rongeured away on each side with angular lamina forceps (Fig. 242),

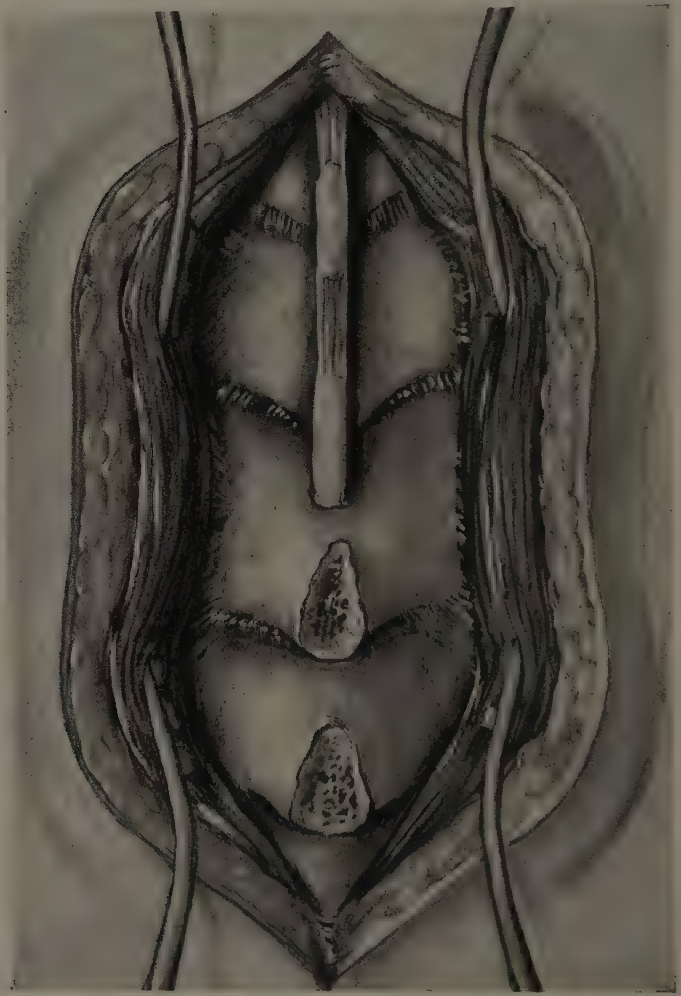

FIG. 241. Laminectomy. Two of the spinous processes have already been removed with rongeur forceps.

beginning below and working upward until the laminae of one or more vertebrae have been removed. By this means the dura, covered by a thin layer of fat, is exposed.

As soon as the removal of bone is completed, the soft tissues all around are covered by strips of moist cotton (Fig. 243).

If the dura is to be incised, two fine silk traction sutures are passed through that membrane, care being taken not to injure any structures within the dural sac. If the cord is pushed backward by a growth in front of it, or if the cord is swollen and the dural sac very tense, the needle carrying the suture must be passed very cautiously. Traction is now made upon the sutures, and with a fine scalpel, a small incision is made in that membrane, care being taken not to incise the arachnoid underneath. A grooved director is then

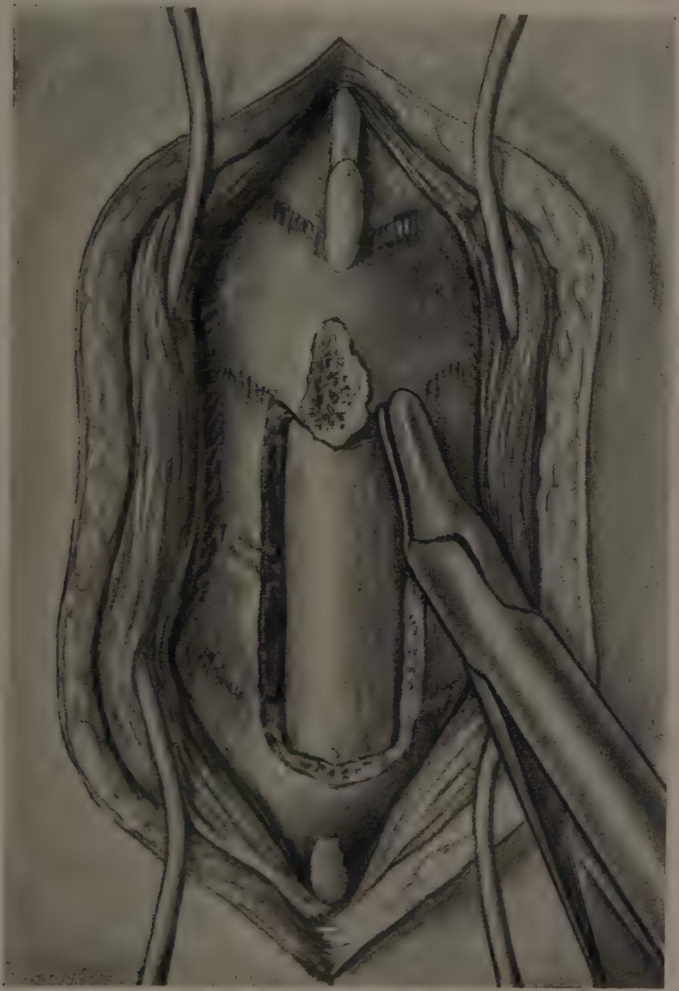

FIG. 242. Laminectomy. Showing the method of removing the laminae with angular rongeurs.

passed into the small opening in the dura (Fig. 243). With slight upward pressure on the grooved director, the dura is split upward and then downward. Fine mosquito forceps now grasp the cut edges of the dura or traction sutures are inserted (Figs. 244, 245).

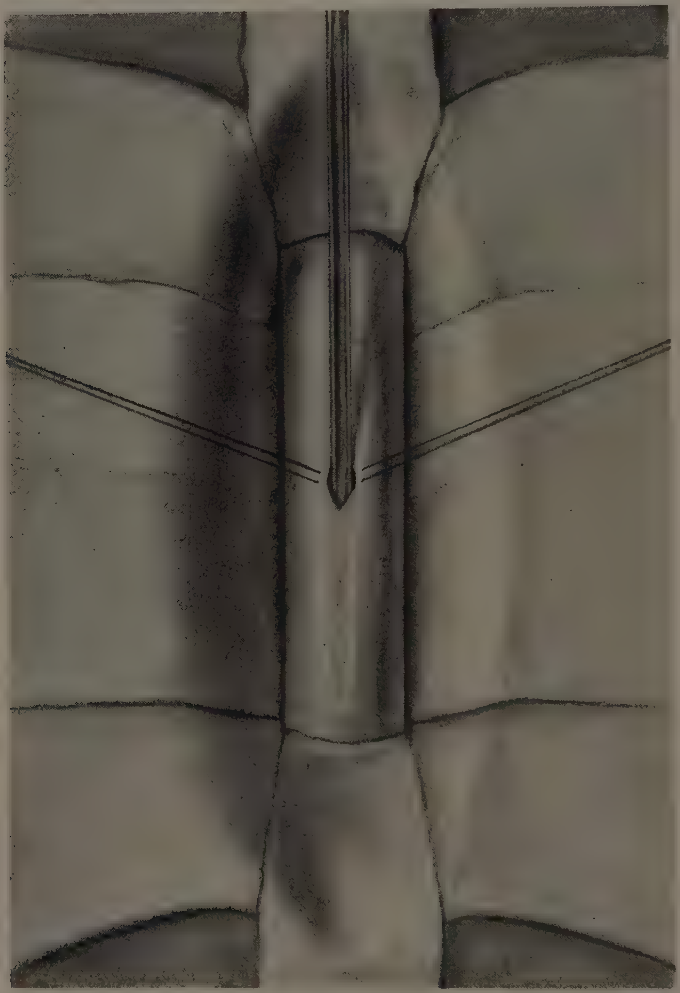

FIG. 243. Laminectomy. After the dura has been exposed and cotton wall-offs are in place. Traction sutures holding the dura. A grooved director has been passed through a small incision in the dura without injuring the arachnoid, and the dura is about to be incised.

The appearance of the various structures that are exposed by the laminectomy and dural incision will vary with the nature of the disease for which the operation is performed. These have been discussed in other chapters.

The amount of bleeding from the soft tissues and bone is seldom very great excepting in angioma of a vertebra, but distinct hyperemia of the soft tissues and bone is frequent in large extradural tumors and in extradural inflammatory processes. In operations for intradural cord tumor, the oozing from many small vessels during the incision of soft parts and removal of bone may make one suspect

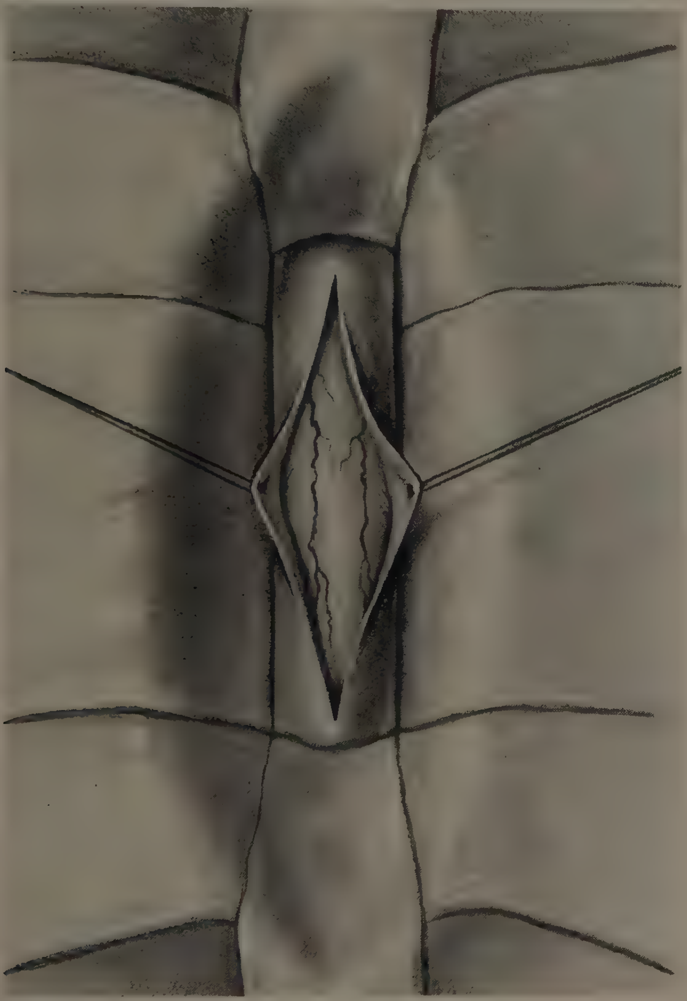

FIG. 244. Laminectomy. The dural edges held open by a traction suture and the cord exposed.

that one is dealing with a meningioma rather than a perineurial fibroblastoma.

The dura is generally covered by a small amount of loose fatty tissue, which can be easily wiped aside by gentle manipulations with a small cotton sponge. The appearance of the dura will often give information regarding the conditions inside that membrane. Pulsation of the dura with the heart beat is generally observed only in the cervical region, but even in that region the absence of pulsation

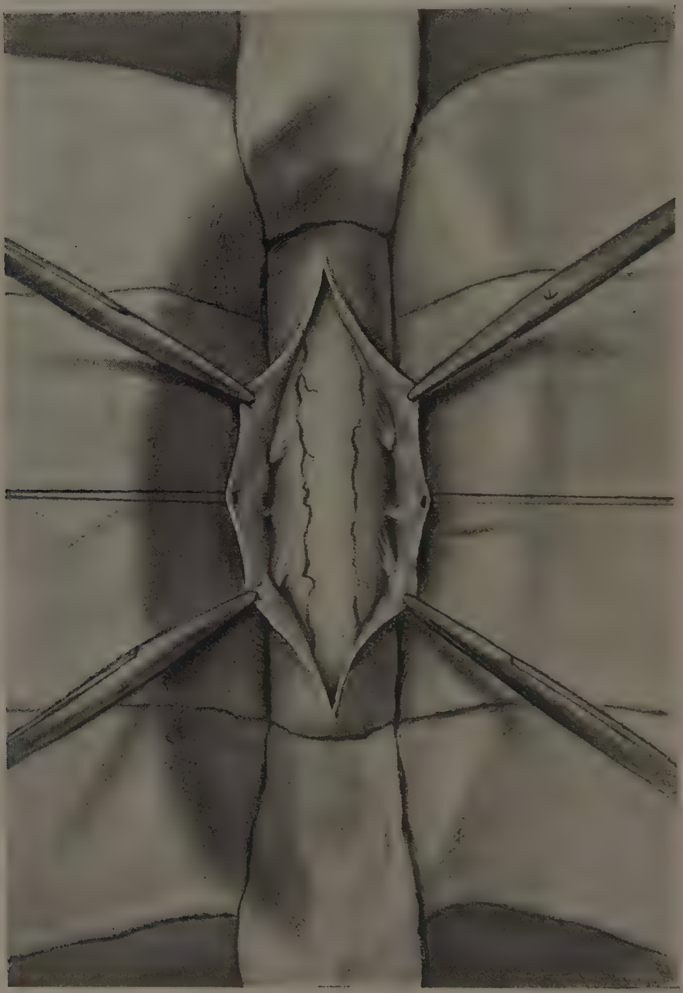

FIG. 245. Laminectomy. The cord exposed by traction on the edges of the cut dura with mosquito forceps.

has little significance. Aside from any gross abnormality of the dura, such as is seen in extradural growths and inflammatory processes, there is usually little to be observed when that membrane is exposed.

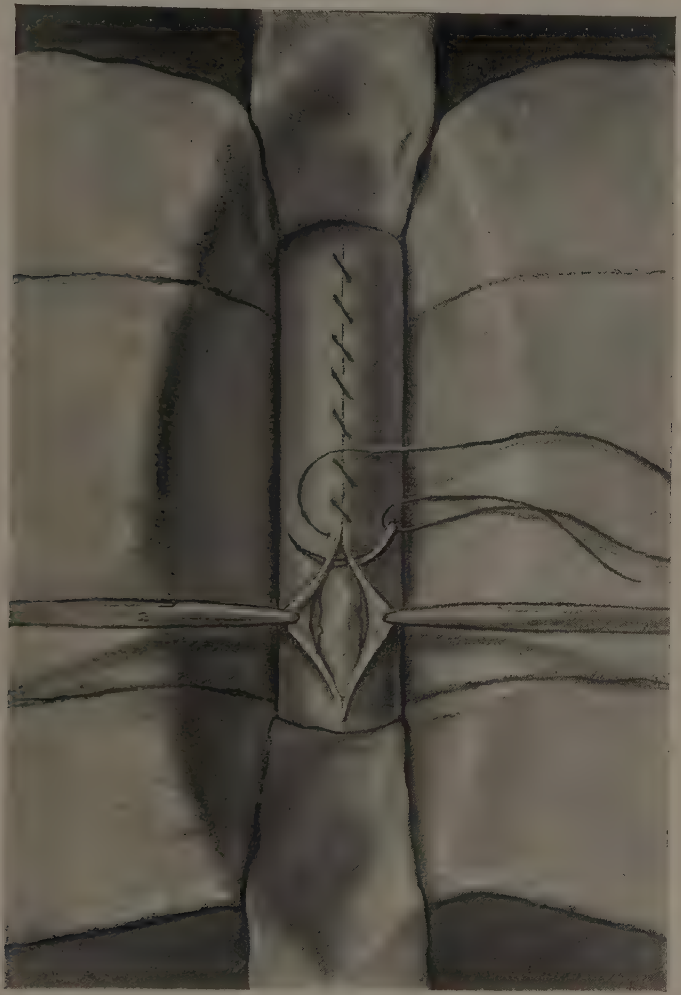

FIG. 246. Laminectomy. The dura is being closed by a continuous suture of fine black silk.

It may appear distended and tense when the spinal cord is much enlarged, or there may be a localized bulging in tumors on the posterior surface of the cord. Sometimes, when the dura is thin (more especially in the lumbar region), a pathological process such as a new growth can be vaguely seen before the dural sac has been opened.

After the dura has been incised without injury to the arachnoid, changes in that membrane (cloudiness, adhesions, tumor adherent to the arachnoid) can be seen. Enlarged vessels on the surface of the cord can often be recognized before the arachnoid sac has been opened.

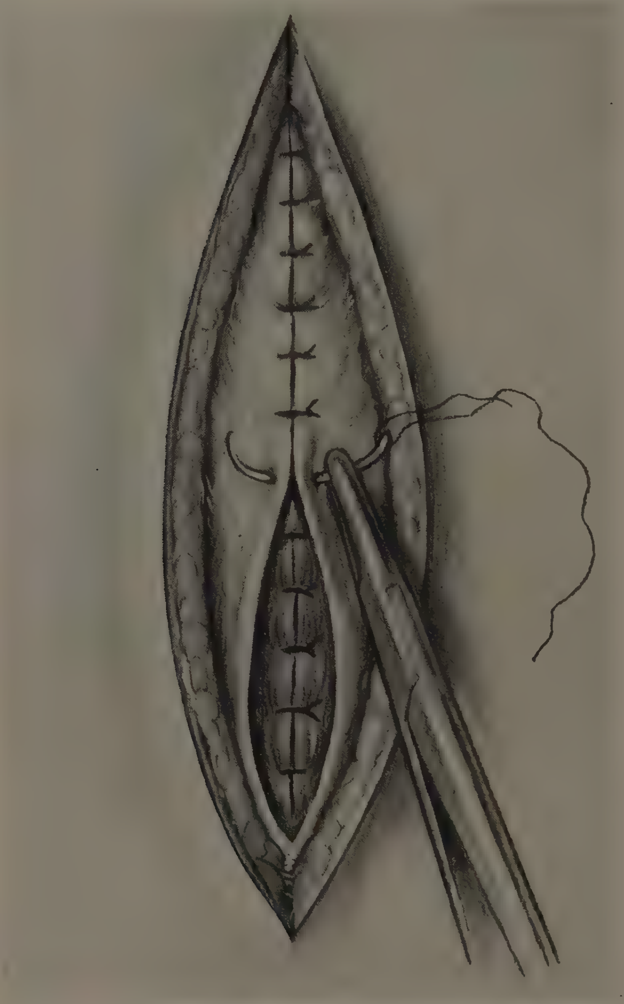

FIG. 247. Laminectomy. The muscles have been closed by interrupted sutures of catgut; the edges of the fascia are being united by interrupted sutures of chromicized catgut.

After the arachnoid has been incised, the spinal cord is at once visible and changes in its color or size, dislocations to one or other side, abnormalities of the posterior spinal vessels, and the like, can be seen.

After the intradural manipulations have been completed, the dura is closed with continuous or interrupted sutures of fine silk, a small amount of saline solution being injected beneath the dura before the last suture is tied (Fig. 246). The entire wound is then thoroughly washed out with warm salt solution and the cotton wall-offs and automatic retractor removed. The muscles are united in layers by interrupted stout catgut sutures. In the cervical and lumbar regions three layers, and in the thoracic region two layers, of muscle sutures are required. The edges of the superficial fascia are then united by interrupted sutures of chromicized catgut (Fig. 247) and those of the skin by interrupted sutures of fine silk. A large, firmly fitting, dry dressing is fixed on the back by strips of adhesive plaster, over which more gauze and a tight bandage are applied.

When the laminectomy has been done in the cervical region, the head is included in the bandage, a long, well-padded splint, extending from the midthoracic region and projecting beyond the head, being incorporated in the bandage. In many cases it is sufficient to immobilize the head by a broad collar of pasteboard or of plaster of paris.

When the operation is performed in the lower part of the spine, soiling of the dressings by urine or feces is prevented by covering the lower part of the dressings with rubber tissue, fixed to the skin just above the buttocks by a broad strip of adhesive plaster.

POSTOPERATIVE TREATMENT

An individual who has had a laminectomy should always be placed on an air or a water mattress after the operation and kept on it for from ten days to two weeks. The head should be raised on a pillow, and the patient should be kept on the back for one to two days. *Hot water bags should never be used.* In fact, those who do spinal operations should have a standing nursing rule that hot water bags should never be used on a patient with a spinal disease or after a spinal operation.

I am accustomed to keep the patient on liberal doses of urotropin for from one to two weeks, or longer if continued catheterization is necessary. Many of the patients have to be catheterized for a number of days or weeks after the operation. The frequency of catheterization depends upon the amount of urine in the bladder; some patients need be catheterized only every eight hours; in others, the urine has to be drawn off every four to six hours. The guiding rule should be that the bladder should never be allowed to become overdistended, because overdistention favors cystitis. Patients are usually constipated and the bowels must be moved by enemas or laxatives. When the laminectomy is performed in the lower thoracic region, many patients suffer from abdominal distention for a number of days—a distention which may not be relieved by effectual enemata. This distention disappears within a few days, and in the meantime the patient is usually made most comfortable by liberal doses of morphine and atropine.

The dressings are left undisturbed for from seven to ten days and the patient is first allowed to sit up two weeks after the operation. Massage, electricity, and exercises of the affected limbs are then begun.

The danger of the operation of laminectomy per se is very small, and if the general condition of the patient was satisfactory before the operation and no complication occurs as a result of the special procedures used, the mortality of the operation should be very small.

CHAPTER XVI

NONOPERATIVE AND OPERATIVE PROCEDURES FOR THE RELIEF OF INTRACTABLE PAIN

OF LATE YEARS, THERE HAS BEEN A NOTICEABLE TENDENCY TO HAVE RECOURSE TO SECTION OF THE ANTEROLATERAL TRACTS IN many persistent painful conditions, without a preceding attempt to afford relief by such procedures as roentgen therapy or paravertebral injections. Some of my patients were relieved by well-directed roentgen therapy, others by paravertebral injections of dilute alcohol. The latter procedure has a wide field of usefulness, but the injections require an exact technique and will accomplish the desired result only if given by those who have trained themselves in the technique.

INTRA-ARACHNOID INJECTION OF ALCOHOL

Another nonoperative procedure by which pain may be relieved, is the intra-arachnoid injection of alcohol. The first reports on the use of intraspinal injection of small amounts of absolute alcohol for the relief of intractable pain were published by Dogliotti (1931), and the procedure has been extensively used. Experience soon showed that patients may be completely relieved of pain by this method, but that there are distinct dangers attendant on its use. The main disturbance that may be produced is loss of control of the bladder and bowels, which may last for a few days or weeks but has persisted for as long as seven to ten months. Other spinal cord symptoms (loss of reflexes and of sensation, diminution of power) have been observed. This is not surprising because histological studies (Alpers and others) have shown that degeneration of nerve roots and secondary changes in the spinal cord may follow a spinal subarachnoid injection of alcohol. It may be that in many of the patients, too large a dose was injected. It may also be that the use of 70 per cent, instead of absolute, alcohol is advisable. We have used subarachnoid injections of 70 per cent alcohol in a number of instances of intractable pain due to metastatic malignant disease, and have seen only very temporary disturbances referable to the spinal nerve roots or the spinal cord. In about one-third of the cases, the patients were either entirely relieved of their pain, or were improved.

The injection should be made between the second and third or the third and fourth lumbar vertebrae, and no more than 1 cc. should be slowly injected. The hips of the patient should be kept higher than the trunk, so that the alcohol is quickly diffused in the spinal fluid.

The method undoubtedly has its dangers and should be used only in patients with intractable pain from primary or secondary malignant disease, in whom the pain is in the lower limbs or the pelvis. The relief may last for a number of months and the injection may be repeated. The dangers attendant upon the procedure should always be explained to the patient or to his family.

Another conservative procedure in the treatment of severe and intractable pain from malignant disease must be mentioned. In rare instances one sees a patient with malignant disease of the spine, who is suffering severe pain which cannot be relieved by drugs, and who already has the symptoms and signs of a complete transverse lesion of the spinal cord. In such a patient, it is justifiable to produce a transverse lesion at a higher level by the injection of a small quantity of alcohol into the spinal cord. The injection should be made only below the first thoracic segment of the spinal cord and the patient may be completely relieved of the pain for the remainder of his or her life.

DIVISION OF POSTERIOR SPINAL ROOTS

In certain localized painful conditions (e.g., after vertebral trauma, in persistent postherpetic or amputation stump neuralgia, causalgia) relief of intractable pain may be obtained by division of posterior spinal roots. This operation was first suggested by Dana and carried out by Abbe. Rhizotomy was once highly recommended, but the operation is now rarely performed because better surgical procedures have been devised. In a considerable number of the patients in whom posterior roots were divided for pain, the relief was only temporary or there was no relief. In many of these patients, an insufficient number of roots were divided, either because of the uncertainty as to how many roots should be cut, or because the surgeon feared that too great a relaxation of muscles or too extensive loss of sensation might result.

Rhizotomy is a procedure which may be performed only when there is a very localized root pain, and in these patients relief may often be obtained by paravertebral injections. Ordinarily, in order to be certain of a result, four to seven posterior roots have to be divided and this means an extensive laminectomy. Even under these circumstances, one cannot be certain that complete relief from pain will be obtained. For the same reasons and because pain was rarely relieved, the division of posterior roots for the severe gastric crises of tabes is no longer performed. However, every one who has performed a number of rhizotomies for pain, will have seen complete or almost complete relief in perhaps 20 per cent of the patients, and numerous successful cases have been reported. The difficulty with the operation is that, no matter how many posterior spinal roots have been resected, one can never be certain that the patient will be relieved of his pain or that the pain will be substantially alleviated. The uncertainty of the operative results have therefore brought it about that rhizotomy for pain is now done comparatively rarely.

The technique of division of posterior spinal roots is a simple one. After the laminectomy has been performed and the dura incised and its edges retracted, the root to be divided is raised with a small strabismus hook; it is first crushed in two spots with a fine mosquito forceps in order to crush any small blood vessels, and then divided with fine scissors. It may be sufficient to crush the root with a forceps instead of actually dividing it. By this means bleeding is prevented.

When roots of the cauda equina are to be divided, it is more difficult to separate the posterior from the anterior roots and to identify the desired roots, unless the roots are exposed near their origin from the lumbosacral cord. The first lumbar posterior root rests on the fork of the dentate ligament (Fig. 3), and an appreciation of this anatomical arrangement is of value for the identification of the adjacent posterior roots.

SECTION OF THE ANTEROLATERAL TRACTS

This operation was proposed by Spiller and first performed by Edward Martin in 1911. The procedure is based upon the fact that afferent impulses for pain pass up in pathways in the anterolateral columns of the spinal cord.

The fibers which transmit pain and temperature sensibility enter the spinal cord in the posterior spinal roots and cross to the opposite side to pass in a cephalad direction in the anterolateral white column. In the cervical and thoracic (and possibly in the lumbar) regions, the crossing only requires one segment (Foerster). According to Foerster and Gagel, pain sensation is carried not only by the crossed anterolateral column but to some extent by the homolateral columns, the peripheral zones of the dorsolateral columns, the long ascending tracts of the posterior columns, the gray horns and the sympathetic system. This is sufficient to explain the fact that sometimes, although the spinothalamic tract has been completely sectioned, some pain sensibility remains, and that months or years after the division of an anterolateral tract, a certain degree of pain sensibility may return, but a normal degree of pain sensibility is never regained.

After section of *one anterolateral column*, there is analgesia in all segments on the opposite side of the body below the level of the section. This analgesia involves skin, tendons, muscles and fasciae, and the bones. By fine tests, it can be shown that pain sensation is altered to a certain degree homolaterally and that tactile sensation is also slightly impaired (Foerster).

In many cases of unilateral chordotomy, temperature sense is lost as well as pain sense, but there are many instances in which the ability to recognize hot and cold is not lost entirely and temperature sensibility may be only slightly impaired. This led to the conclusion (Foerster, 1927; Tilney and Elsberg, 1928; and others) that possibly the fibers for temperature run separately in the most dorsal part of the anterolateral tract.

After *bilateral section* of the anterolateral tracts, both pain and temperature sensibility are lost in the areas below the segment through which the section has been made. The loss of sensation involves all tissues including the viscera. Temporary retention of urine occurs regularly after bilateral chordotomy. In males, the ability to have sexual intercourse and orgasm is lost.

As there is a lamellation of the fibers in the anterolateral tracts according to limbs and parts of limbs and the fibers for the upper limbs lie more ventral and internal than those to the trunk and lower limbs, a section of the anterolateral tract may not be made deep enough to divide the fibers from the upper limbs. This explanation has been suggested for the fact that sometimes pain perception has remained after bilateral division of the anterolateral tracts. Foerster believes that the occasional persistence of pain sensibility after chordotomy may be due to the existence of a pathway in the posterior gray horns of the spinal cord. From my personal experience, it has appeared to me that the first explanation is the more probable one, although the possibility that there may be a posterior horn pathway cannot be denied. If, in such a case, a second operation is performed, and the tract sectioned more deeply and more anteriorly, pain sensibility will be lost.

It appears certain that as one progresses from the lower segments of the body upward, the corresponding pain fibers are disposed more and more anteriorly in the spinal cord. It is possible, therefore, by a differential section of the fibers, to eliminate the sensation of pain in the chest and to retain it in the lower extremities (Hyndman and Van Epps, 1939).

Indications for Operation

Section of one or both anterolateral tracts is often the only therapeutic procedure which can be used for the intractable pain of primary or metastatic malignant disease. The operation should be performed only in the patients who will live, in all probability, for at least a number of months. Manifestly, nothing is gained by subjecting a patient who will live only a month to the strain and discomforts of an operation.

Chordotomy may be of value for the relief of other painful conditions, such as inveterate neuralgia after injuries or inflammatory diseases of the spinal cord and nerve roots. Persistent causalgia may be relieved by section of the anterolateral tract of the opposite side. The pain of chronic infectious arthritis has been made to disappear by bilateral chordotomy in the cervical region. The gastric crises of tabes can be eliminated by bilateral section of the anterolateral tracts at or above the third thoracic segment. Pain of thalamic origin has been relieved, in a few instances, by the operation.

Technique of Operation

The operation does not offer any special difficulty to the surgeon experienced in operations upon the spinal cord. The success or failure of the operation depends entirely upon the accuracy with which it is performed, and the gentleness and care with which the tissues are handled.

The laminectomy consists of the removal of the arches of two, or at the most three, vertebrae. After the dural sac has been opened, the arachnoid is also incised and a slip of the dentate ligament seized with fine mosquito forceps and cut at its attachment to the dura. The cord is then gently raised and rotated to expose its anterolateral surface. An area is then selected which is free from visible blood vessels and the arachnoid again carefully incised. The incision in the cord is made with a fine-pointed sharp knife and should penetrate the cord well anterior (at least 2 mm.) to the attachment of the dentate ligament (Fig. 248). The knife should be carried forward parallel to the anteroposterior axis of the cord and should emerge either through or anterior to the anterior spinal root (Fig. 249). The depth of the incision should be from 2.5 to 3 mm. in the thoracic region and from 3 to 4.5 mm. in the cervical region. If the site for the incision has been properly selected, bleeding should not result from the incision in the cord. If the anterolateral tracts of both sides are to be divided, the sections should be made at different levels, ½ cm. to 1 cm. apart.

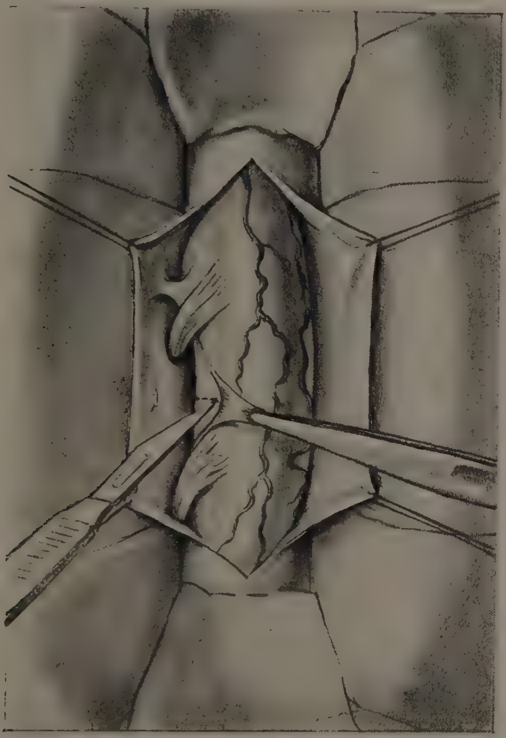

FIG. 248. Laminectomy. Section of the left anterolateral tract; the cord is rotated by forceps grasping the cut end of the dentate ligament. The location of the incision is shown by a broken line. (Enlarged to show details.)

The level at which the laminectomy is to be performed and the tract in which section is to be done will depend upon the segmental areas involved in the pain producing lesion. It should be made at least two segments higher than the area to be made analgesic. The anterolateral tract may be divided at any level of the spinal cord, but high bilateral cervical sections should seldom be done at one operation because of the danger of interference with respiration.

In general the loss of pain sensibility will be greater during the first few days after the operation than subsequently, and this is especially the case if the section has not been made deep enough. If the section has not extended far enough anteriorly, the level of the loss of pain may retract toward the area of permanent loss after a few weeks.

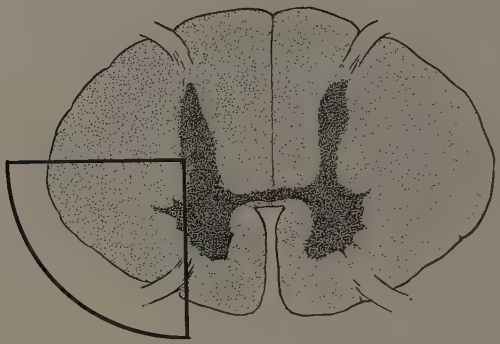

FIG. 249. Transverse section of the spinal cord showing the area involved in section of one anterolateral tract (diagrammatic).

One advantage in performing the operation under local anesthesia is that after the section of the anterolateral tract, the extent of loss of pain sensibility can be controlled at the operating table. This may avoid the disagreeable experience of discovering either that the pain has not been eradicated or that the loss of pain sensibility does not extend high enough, so that a second operation is necessary. The patient does not experience any pain when the spinothalamic tract is cut, but when the operation is performed under local anesthesia, the posterior roots in the exposed area should be anesthetized with novocain as they are very sensitive to handling.

Girdle pain is very frequent after the operation and is proportional to the degree to which the posterior spinal roots have been manipulated.

It is hardly necessary to state that if a unilateral section of the anterolateral tract is done for pain on one side of the body, the tract of the opposite side must be divided.

The operation must often be performed in patients who are worn out by intractable pain or debilitated by a malignant disease, but it is generally well borne. There should seldom be any marked loss of power of the limbs, although a temporary weakness may occur, as after any laminectomy with manipulation of the cord tissue. Retention of urine is frequent, especially after bilateral section of the spinothalamic tracts; it may last for a few days or weeks, but it has persisted for many months and in some cases it has been permanent. After cervical chordotomy, a Horner syndrome is usually observed.

MYELOTOMY OF THE COMMISSURE FOR PAIN IN THE UPPER EXTREMITIES

In 1934, Putnam suggested a new operative method of treatment for pain in the upper limbs, which he had used with success in several patients with pain in *both* upper extremities. By means of a spinal instrument which was inserted between the posterior columns without injury to the posterior spinal vessels, the commissure was divided from the upper cervical to the upper dorsal region. After the operation, there was loss of pain and temperature sensibility over the chest and both upper limbs. The pain was relieved in two of three patients. Since his paper was written, Putnam has operated upon another patient with success. Cases in which this operation may be indicated are rare, and further experience will be necessary before one can judge the efficacy of the method and its advantages or disadvantages.

A somewhat similar operation had been proposed by Greenfield and carried out by Armour (1927) in one patient with severe gastric crises. A mediolongitudinal incision of about 1 inch in length was made at the level of the eleventh and twelfth dorsal and first lumbar segments. The patient succumbed from pneumonia a few days after the operation.

CHAPTER XVII

OPERATIVE PROCEDURES FOR THE RELIEF OF SPASTIC CONDITIONS AND OTHER MOTOR DISTURBANCES

DIVISION OF POSTERIOR SPINAL ROOTS FOR THE RELIEF OF SPASTICITY

IN 1908, FOERSTER SUGGESTED THAT IN SPASTIC CONDITIONS OF THE LIMBS DUE TO LOSS OF CORTICAL INHIBITION, RELIEF WOULD FOLLOW the division of the appropriate posterior spinal roots. The suggestion was based on the fact that muscle tone depends upon a reflex arc whose afferent limb carries the fibers which transmit impulses from the periphery to the motor cells in the anterior gray horns of the spinal cord. The efferent messages sent to the muscles are controlled by inhibitory fibers from the brain in the pyramidal (and extrapyramidal) pathways.

Following this suggestion by Foerster, rhizotomy was performed by a large number of surgeons in a variety of conditions: congenital spastic paraplegia and diplegia (Little's disease), spinal spastic paralysis, spasticity associated with multiple sclerosis, myelitis, and old traumatic lesions of the spinal cord.

The first reports of the results of rhizotomy for spasticity were encouraging, but as more experience was gained, the reports were less and less favorable. Although the operation is founded on a sound physiological principle, complete restoration of function could never be expected, and a critical analysis of the results of the operation would have to be based upon the degree of improvement that occurred, that is, whether a patient bedridden by the spasticity could be made to walk again, or whether a crippled gait was greatly improved.

Considerable of the improvement that occurred after the operation, was found to be due to the thorough mechanicotherapy and physiotherapy that was given after the operation. Therefore, the true value of rhizotomy for spasticity could only be appraised if the operation was performed in patients who had derived the maximum amount of benefit from massage, muscle re-education, tendon lengthening, and orthopedic appliances.

Furthermore, it was soon shown that after cervical root section, spasticity of the upper limbs was often little, if at all, improved, and that recurrence of spasticity was frequent. This may have been due to the fact that too few posterior roots were resected, because of the fear that too great a sensory disturbance would result. However, the operation was soon limited to patients with spasticity of the lower extremities.

Little can be gained by the operation, if the spastic muscles are paretic or completely paralyzed, for the only result will be a paralyzed instead of a spastic limb. However, even under these conditions, the operation may be done in markedly nonprogressive lesions of the spinal cord, when the patient's limbs may be relaxed and straightened out, instead of being flexed, with the legs against the thighs and the thighs against the abdomen.

INDICATIONS AND CONTRAINDICATIONS

Division of sensory roots for the relief of spasticity of the lower extremities may be done only in stationary lesions or in those in which progression is exceedingly slow, such as Little's disease in particular, and spastic conditions remaining after a spinal trauma or a spinal disease that has run its course. In congenital spastic paraplegia, the operation should not be performed if the mental condition of the child is such that muscle re-education after the operation will be impossible. If the patient's mental state is sufficiently good, posterior root section may be tried in spasticity of the lower limbs, provided the patient's family understand that the chances of improvement are small and that at least one to two years of mechanicotherapy and re-education will be necessary in order to obtain the maximum benefit from the operation. In several young patients with Little's disease, I have seen very great improvement lasting for a number of years; but in the majority of the cases, the result did not justify the means. The operation will not influence athetoid movements, and section of posterior roots is contraindicated if there are athetoid movements associated with spasticity.

Technique

The most important technical detail in the operation of rhizotomy for spasticity is the selection of the roots to be divided. I have been accustomed to cut two of three roots from which a given group of muscles derives its sensory supply. If too many roots are divided, too great a relaxation of the muscles may follow so that the patient may be left with more or less flaccidity which may result in a limb as useless as when it was spastic.

In the lower extremities, the muscles most seriously affected by spasm are usually the adductors of the thigh, the hamstrings, and the calf muscles. Ordinarily, it is necessary to divide the posterior roots of L 1, L 2, L 3, L 5 and S 1, or L 2, L 3, L 5 and S 1. It is usually advisable to preserve L 4 root as this root has to do with the muscles that extend the leg at the knee. The exact roots that must be cut must be decided upon after a careful investigation of the muscles or muscle groups that are especially involved in the spasm.

The cases in which section of posterior roots for spasticity is indicated are not many, and the operation is now rarely performed. However, the procedure has been of value in carefully selected cases. I have had a number of satisfactory results in patients with spasticity of the lower limbs resulting from traumatic irremediable lesions of the spinal cord. Bedridden patients were made much more comfortable by the relaxation of limbs drawn up against the body by spasm, and patients with marked spasticity of one lower limb were able to walk without crutches or support after the division of carefully selected posterior spinal roots.

DIVISION OF POSTERIOR SPINAL ROOTS AND OF THE SPINAL ACCESSORY NERVE FOR SPASMODIC TORTICOLLIS

The etiology of spasmodic torticollis is still little understood and the significance of the psychogenic factor is still uncertain. Foerster believes that many cases are of striatal origin, but it is appreciated that the movements are always exaggerated when the individual is fatigued or excited, or more self-conscious as when in public places or in the physician's office.

Many muscles are concerned in the movements of spasmodic torticollis, especially the sternocleidomastoid, trapezius, splenius capitis, splenius cervicis, semispinalis capitis, longus capitis, the scaleni, the small muscles between the atlas, axis and occipital bone (rectus capitis and obliquus anterior and posterior).

Different degrees of *bilateral* involvement of corresponding muscles or involvement of some muscles of one side and of other muscles of the opposite side are frequent. It is probably rare that the muscles of only one side of the head and neck are affected.

Most of the muscles involved in spasmodic torticollis receive their innervation through the upper four cervical roots, although some of the muscles are supplied also through lower cervical roots, and the sternomastoid and trapezius mainly through the spinal accessory nerve.

Many efforts have been made to relieve spasmodic torticollis by surgical means. For years, attempts were made by division of the affected muscles or by section of their motor nerve supply (Stoffel operation), but most of these procedures have been given up. In 1891, Keen attempted to relieve the condition by section of the posterior divisions of the upper three cervical nerves. In 1915, basing his proposal on the efforts of Foerster to relieve the spasticity of Little's disease by section of the posterior spinal roots, Taylor performed a unilateral intraspinal division of the upper four posterior roots for spasmodic torticollis. The patient was much improved.

In 1924, McKenzie described an operation in which he divided both anterior and posterior roots as well as the spinal accessory nerve on the same side.

Finney and Hughson (1925) were the first to perform a bilateral operation, in which the posterior divisions of the upper three cervical nerves on both sides were divided outside of the vertebrae and both spinal accessory nerves were divided. In 31 cases, some of which had been followed for twenty years, 12 patients were cured and 16 were improved.

The excellent results obtained by Finney, led other surgeons to perform similar operations, in which the sensory or sensory and motor roots of the upper three cervical roots and one or both spinal accessory nerves were divided (Dandy, 1929; Frazier, 1930; and many others). On account of the fact that muscles on both sides of the body are usually involved, the operation should be bilateral.

Modern operations for the relief of spasmodic torticollis have been limited to (1) bilateral division of the first,* second and third cervical posterior spinal roots and division of the spinal accessory nerve on one or both sides; (2) bilateral division of the upper three anterior spinal roots and the spinal accessory nerves; (3) bilateral division of both anterior and posterior spinal roots and spinal accessory nerves.

There is some difference of opinion regarding the question whether it is sufficient to divide only posterior spinal roots or whether, as suggested by Dandy, both anterior and posterior roots should be cut, or whether, finally, only the anterior roots need be divided. My own experience has been with division of the posterior roots and of the spinal accessory nerves only. The area of loss of cutaneous sensibility is sometimes surprisingly small; in other instances, it involves a large part of the head and neck. Further experience may show that it is sufficient to divide only the anterior roots. On account of its relation to the phrenic nerve, the fourth cervical root should not be cut.

As far as the spinal accessory nerves are concerned, they may be divided at the same operation at which the laminectomy is performed, or one to two weeks later. The little operation can be satisfactorily performed under local anesthesia or with no anesthesia. It may be sufficient to divide the spinal accessory nerve only on the side on which there is the marked spasm of the sternomastoid, but not rarely a bilateral operation is necessary. The reason why it is advisable, excepting in the most severe cases, to divide the spinal accessory nerve or nerves at a second operation, is that sufficient relaxation of the muscles may be obtained without the cutting of the spinal accessory nerve. On account of the ease with which the nerves can be cut through a small incision along the anterior border of the sternocleidomastoid muscle, it has been found preferable to divide the spinal accessory nerves from in front rather than to attempt to expose the nerves by removal of part of the foramen magnum.

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* There is usually no posterior division of the first cervical root.

Operative Technique

Little need be said regarding operative technique. The position of the patient is that employed for suboccipital craniotomy or for high cervical laminectomy, and the exposure of the cervical cord up to the foramen magnum offers no special difficulty. Unless the operator plans to divide the spinal accessory nerves within the cranial cavity, the posterior margin of the foramen magnum need not be removed.

As soon as the dura has been incised, the posterior roots are readily seen and easily identified. Whether the roots are divided or are simply crushed with a fine clamp is a matter of individual preference.

In order to expose the anterior roots, it is usually necessary to divide one slip of the dentate ligament and gently raise the cord by traction on the ligament.

The first result of the division of the upper three sensory or motor roots or both sensory and motor roots, is to make the head somewhat insecure, so that the patient feels a more or less marked lack of ability to support the head. This weakness is gradually overcome. Some of the muscles retain part of their nerve supply after the operation and therefore function to a restricted degree. These muscles are adequate to take care of the required movements of the head and neck for all practical purposes. After several months, the patients no longer complain of lack of muscular support of the head and are able to move the head and neck freely.

After the operation, the spasmodic movements may not cease entirely, but with exercises, the movements that persist will disappear within a few weeks or months. The importance of postoperative training in eliminating any remaining muscular incoordination must be emphasized.

As already mentioned, the sensory loss may be extensive over the head and neck, or it may be limited to only a part of the neck.

Section of Extrapyramidal Motor Tracts for Athetosis and Dystonia

A few years ago, Putnam performed a series of operations in which he divided the extrapyramidal motor tracts for athetosis and dystonia, with resulting improvement in 4 of 5 patients. In Putnam's own words: "The extent of the improvement is difficult to gage objectively, but it is perhaps significant that three of the patients have of their own accord requested a further, more radical operation, and a fourth has requested operation after seeing its effect on her sister. . . . Section of the anterior group of tracts is apparently innocuous. How deep the incision should be made anteriorly, and whether the prepyramidal should also be severed, must be determined by further experience." Putnam's conclusions were:

1. "In two cases of familial dystonia musculorum, one of bilateral choreo-athetosis following an acute infectious disease in infancy, and one of unilateral athetosis following an acute infection in adult life, a decrease in the abnormal movements has been brought about by operative section of the prepyramidal and anterior quadrant marginal fibers, or the latter alone, in the cervical cord.

2. "The operation was followed by mild signs of pyramidal tract defect in one case and hemianalgesia in another. There were no permanent ill effects from the operation and (except in one case) no sensory loss.

3. "In a case of juvenile paralysis agitans following encephalitis, the symptoms were neither relieved nor aggravated by the operation."

The number of cases was too small to allow more than general conclusions in regard to operative indications and technique. Efforts like this are certain to help us in the search for a method of treatment of some cases of dystonia and athetosis. Even extensive root sections are useless, as I found many years ago.

In this connection one might mention the recent investigations of Bucy, on the effect of ventromedial chordotomy on muscular spasticity in man. In 3 patients suffering from congenital spastic paraplegia or quadriplegia, one or both anteromedial columns of the spinal cord were sectioned in the upper thoracic region. No permanent reduction of spasticity resulted.

MYELOTOMY IN THE TREATMENT OF SYRINGOMYELIA

At the International Congress held in London in 1913, I described an operation which I had performed in several cases of syringomyelia. This consisted of laminectomy, and incision and drainage of a cystic cavity in the spinal cord. One of the patients operated on had been bedridden, had lost much power in the upper limbs, and had a spastic paraplegia. After the myelotomy, performed in the cervical region, the power in the upper limbs became greatly improved and the spasticity and loss of power in the lower limbs much less. The improvement lasted for over two years. In the second patient, the improvement was also marked, and lasted for three years.

In 1926, Puusepp described a similar operation, and a large number of cases have been reported in recent years. In a number of the cases, there was considerable improvement which lasted for from one to seven years. Putnam and Munro (1931) recorded 4 cases and referred to a large number of operations performed by others [Foerster, Jirasek and Vitek, Zeno and Cames, Oppen (6 cases), Lafora, Schmieden, Gorsky, Ellmer, Kapis, Juzelevsky (14 cases), Kuttner, Sicard and Robineau (2 cases)]. The most recent publication on the subject is that of Frazier and Rowe, who collected from the literature 14 cases which had been observed for more than one year and added 2 of their own.

The results of the operation appear to have been beneficial in a large proportion of the patients, but many of the reports were of single cases that had been improved, and single cases in which the patients were not benefited, may not have been reported.

Improvement following operation has consisted in relief of pain, decrease of spasticity in the lower limbs, and improvement in sensory and motor disturbances of the upper limbs.

The patients with syringomyelia in whom an incision into the cord and drainage of a cystic cavity is justified, are, according to my belief, those cases in which it is possible to conclude that the symptoms are due in great part to pressure upon the cord by the collection of fluid within the cord substance. These individuals, in addition to sensory disturbances and muscular atrophy in the upper limbs (and especially of the hands) have more or less spasticity in the lower limbs. In patients with this clinical picture, the operation will probably be followed by considerable improvement. In patients who did not present such symptoms, I have seen little practical benefit from the incision in the cord.

Operative Technique

The technique of the operation is a simple one. After exposure of the enlarged cord by laminectomy and incision of the dura, the cord is aspirated with a fine hypodermic needle. As soon as fluid is withdrawn, the needle is removed, and an incision made in one posterior column just lateral to the posterior median septum. The incision is slowly deepened until the cystic cavity is opened, and is then enlarged upward and downward until it is about 2 cm. in length. With the retraction of the edges of the incision in the cord, it is usually possible to see the walls of the cavity. I see no advantage and possible danger in attempting to measure the length of the cavity by exploration with a catheter.

Some surgeons have maintained drainage by the use of a small gutta percha drain (Frazier), others by linen thread or piece of dura, and still others by suturing or clipping arachnoid or dura to the wall of the cavity. Although there are several reports that at a second operation, the opening was found to be closed by adhesions, I am not at all certain that drainage of the cavity by any material is necessary if the incision in the cord has been of sufficient length and the lips of the cord incision have been left perfectly dry.

At the conclusion of the operation, the dural incision should be closed by suture.

Although a considerable number of operations for syringomyelia have already been performed, there is as yet no agreement as to the exact type of case in which operative intervention is justified. The future must show when myelotomy for syringomyelia is indicated, how much to expect from the procedure, and how often and how great the improvement.

The only other method of treatment is by x-ray therapy, and in spite of many reports, it is not certain how much, if any, benefit may be expected from radiation treatment.

APPENDIX

MULTIPLE TUMORS OF THE SPINAL CORD

IF METASTASES FROM PRIMARY MALIGNANT DISEASE (IN WHICH THERE MAY BE NUMEROUS SECONDARY EXTRADURAL, EXTRAMEDULLARY, or intramedullary growths) are excluded, multiple tumors of the spinal cord, spinal membranes, or nerve roots occur most often in neurofibromatosis (von Recklinghausen's disease), myeloma, chordoma, and Hodgkin's disease. Sarcoma of the spinal meninges may be multiple and this variety of tumor has a tendency to extend along the pia mater. A case of multiple "endothelioma" of the dura was reported many years ago, but the growths were probably pial sarcomas. Some of the cases that have been published as instances of multiple sarcoma may have been multiple neurofibromas of von Recklinghausen.

Multiple tumors of the central nervous system, in which there is a single spinal cord tumor and one or more brain tumors are not infrequent. In von Recklinghausen's disease there may be growths in the cerebello-pontine angle of one or both sides and growths on the sciatic or other peripheral nerves. In rare instances of which I have seen one example, there are several dermoid cysts—one in the posterior cranial fossa and another on the surface of the spinal cord. After the incomplete removal of a posterior fossa medulloblastoma, tumor cells may be implanted in the subarachnoid space and form secondary growths on the surface of the thoracic or lumbar cord (page 304 *et seq.*).

The tumors in multiple neurofibromatosis most often involve the cervical spinal roots or the roots of the cauda equina. Antoni (1920) has described a case in which every one of the cervical spinal roots had a neurofibroma on it. Among others von Büngner (1897), Reichmann (1912), and Elsberg (1925) have described multiple growths of the roots of the cauda equina. In my case, the patient also had a sciatic tumor and a tumor in the right cerebello-pontine angle. Most often, there are small tumors on only a few of the cervical or lumbosacral nerve roots. The growths may be intradural or extradural or both, and in rare instances intramedullary. They are usually small in size (1 cm. to 2 cm.) and are usually derived from the posterior roots. Bone changes in the vertebral column are frequent (page 106). Not rarely there is a kyphoscoliosis with a marked gibbus in the thoracic region of the spine. The symptoms and signs of compression of the spinal cord may be slight or marked. They may be due to the marked kinking of the vertebral column, but more often they are caused by growths on the nerve roots which disturb the functions of the roots and make pressure upon the spinal cord. The symptoms are similar to those produced by spinal cord compression by other types of tumor, although occasionally the occurrence of root symptoms at two distinct levels of the cord makes the pattern of the neurological disturbances a more complicated one.

Multiple myelomas are usually extradural and may occur in any part of the spine. They are most often found in the thoracic part of the cord and the disease can sometimes be recognized or suspected from the character of the bone destruction seen on roentgen ray films of the spine.

Multiple myeloma is about twice as frequent in men as in women and occurs in middle age or the later periods of life. Pain, rheumatic in character, is often the first symptom, and almost one-half of the patients have neuralgic disturbances. The degree of interference with cord function depends upon the size and number of the growths. Most of the patients I have seen have had a spastic paraplegia without involvement of the upper limbs. Roentgen ray examination of the spine usually, but not always, shows changes which can be recognized by the expert roentgenologist (page 112). In some of the patients, the typical Bence-Jones bodies are not found in the urine, but there are atypical proteins which have the same diagnostic significance as the former. In two of my patients Bence-Jones protein was not found in the urine on repeated examinations.

Chordoma occurs most often in the upper cervical or in the lumbosacral region. In the former instance, there may be bone destruction also of the base of the skull, and in the latter instance, of the pelvic bones.

In 10 to 14 per cent of cases of Hodgkin's disease (lymphogranuloma) there are lesions in the vertebral column. Spinal cord and nerve root symptoms are produced either by lymphogranulomatous masses in the epidural space by direct involvement of nerve roots, or by secondary softening of the spinal cord due to interference with the blood supply by the tumor masses. The thoracic cord is most often involved—in more than 75 per cent of cases.

The presence of multiple tumors must always be suspected in patients who have the symptoms and signs of compression of the spinal cord and who are known to be suffering from von Recklinghausen's disease or Hodgkin's disease or in whom a diagnosis of chordoma or myeloma has been made from the bone destruction found in roentgen ray films of the spine. From the clinical signs alone it is rarely possible to determine more than the possibility of multiple growth. If the tumors are at different levels, the disturbances in motor power and sensation produced by one of the growths will predominate; if the predominating signs are due to the growth at the higher level, the signs due to the one at the lower can hardly be distinguished from those due to the growth at a higher level. Occasionally, the occurrence of root pain at two levels well separated from each other, has made it possible to make a diagnosis of tumors at two levels, but the finding of two sensory levels—even if they are distinct—is not sufficient evidence upon which to base a diagnosis of more than one tumor.

Unless an individual is known to be suffering from metastases from malignant disease in some other part of the body, the surgeon must be guided by the signs and symptoms of compression of the spinal cord in determining the indications for operative intervention. If more than one growth is found in the area exposed by laminectomy, the attempt should be made to remove all diseased tissue. In neurofibromatosis the growths can sometimes be peeled from the roots; in other cases parts of the roots or several may have to be sacrificed; but one must remember that profound disturbances in function may be produced by division of cervical or lumbar roots.

If neurofibromas have been removed from several roots, the signs of compression of the spinal cord may be entirely relieved and as far as spinal symptoms are concerned, the patient may remain well for many years. Considerable relief may be obtained in other forms of multiple growths by partial removal of one tumor, followed by x-ray therapy. In spite of thorough radiation most of the patients succumb to their disease within a few years, but I have one patient who is fairly well more than five years after the partial removal of a myeloma from the mid-thoracic region followed by radiation therapy. In this case there is x-ray evidence of the disease at another level of the spine. Up to the present, operations for chordoma are only palliative.

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INDEX OF AUTHORS

Abbe, 536
Abbott, 125
Adson, 200, 201, 204, 217, 234, 235, 343, 344, 350
Alajouanine, 469, 508
Albee, 491
Alexander, 511
Allessandri, 519
Alpers, 535
Andrae, 469, 471
Antoni, 26, 214, 215, 233, 240, 248, 368, 369, 373, 382, 407, 494, 497, 554
Aoyagi, 264
Armour, 543
Auerbach, 407, 430
Ayer, 34, 35, 471

Babinski, 140, 374, 385, 396, 415, 435
Bailey, 235, 248, 281, 288, 293, 324, 330, 338, 366, 495
Balint, 204
Balo, 503
Barr, 61, 471
Barré, 397, 398, 435
Bassoe, 324
Beer, 453
Beevor, 161
Bell, 517
Benda, 129
Benedikt, 204
Bickham, 519
Bing, 215
Bircher, 215
Boerner, 214
Bonomo, 519
Borchardt, 214, 519
Bouchard, 264
Brasch, 507
Braun, 453
Bregman, 365
Brewer, 100
Brock, 13, 341, 359, 498
Brodnitz, 407
Browder, 355
Brown, 214
Brun, 453
Bucy, 100, 465, 466, 469, 495
Büngner, von, 554
Burr, 161, 264
Busser, 498
Byers, 161

Cabot, 491
Cairns, 317, 324
Camp, 471
Campbell, 185, 189
Capp, 495
Cavazzani, 202, 203
Cavicchia, 519
Cestan, 507, 508
Charcot, 189, 428, 429
Chiari, 168, 203
Chipault, 491
Chopart, 517
Clarke, 46
Cloward, 100, 465, 466
Cobb, 510, 511
Coburn, 168
Cohn, 129
Collier, 203
Collins, 397
Cone, 27, 167, 177, 267, 270
Cordero, 503
Coughlin, 179
Cramer, 36
Crothers, 160, 161
Cushing, 226, 234, 251, 254, 262, 324, 330, 338, 453

Dacosta, 454
Dailey, 40
Dana, 536
Dandy, 548
Davison, 355, 359, 432
Dawbarn, 518
Déjérine, 11, 140
Denk, 209
Denker, 359, 498
Dercum, 454
Desault, 517
Dogliotti, 535
Domenici, 209
Donald, 168
Doshay, 511
Dubreville, 203
Dyke, 42, 71, 72, 100

Eiselsberg, 453
Eisenhardt, 262
Ellerman, 498
Elsberg, 26, 27, 36, 71, 72, 100, 204, 214, 253, 369, 453, 507, 538, 554
Enriquez, 374
Epps, 539
Ewing, 349

Feil, 48
Feiling, 453
Feinberg, 202
Fincher, 471, 474
Finney, 547
Fischer, 314
Flatau, 14, 210, 365, 369, 436, 451
Foerster, 10, 14, 129, 130, 132, 135, 140, 267, 281, 293, 309, 365, 369, 383, 397, 453, 538, 544
Foix, 34, 41
Ford, 160, 161
Forestier, 112
Francini, 240, 249
Frazier, 511, 548, 551
Fremont-Smith, 40
Friedman, 124, 430
Froin, 41
Fuchs, 383
Fulton, 13

Gardner, 407
Gaupp, 510
Gehuchten, von, 400
Globus, 254, 263
Goldstein, 209
Golgi, 264
Graftdijk, 168
Grant, 27
Gredig, 168
Greenfield, 312, 315, 543
Groff, 323
Gross, 166, 230
Guillain, 197, 508
Guizzetti, 503

Hammes, 496
Hampton, 472, 479
Hare, 27
Harris, 124
Harvey, 264
Hassin, 156, 265
Hawk, 471
Head, 384, 388, 394, 398
Heboldt, 503
Heilbronner, 369, 396
Heister, 517
Henneberg, 403, 408
Henschen, 267, 494
Herzog, 253
Heuer, 214
Hibbs, 491
Hildanus, 517
Hitchcock, 203
Hitzrot, 495
Holmes, 153, 389
Homen, 203
Horsley, 194, 430, 518
Hortega, 267
Horwitz, 124
Hosoi, 348
Hughson, 547
Hutchinson, 45
Hyndman, 539

Jacox, 498
Jarkowski, 140, 397, 415, 435
Jerasik, 209
Josephy, 235, 303

Jumentié, 374, 396, 510
Junghanns, 495, 496

Kahn, 498
Karger, 368, 402
Karplus, 397, 398
Keen, 547
Keiller, 166
Kennedy, 204
Kernohan, 217, 234, 245, 248, 271, 276, 283, 305, 325, 343, 344, 350, 359, 432
Keschner, 355, 432
Klemme, 498
Klippel, 48
Kohler, 507
Kolodny, 166, 179
Kooy, 161
Krause, 430, 453, 510
Krüger, 194
Kümmel, 55
Kyuno, 264

Lambotte, 400
Langley, 371
Lannelongue, 425
Learmonth, 252, 253
Lebert, 263
Leroux, 227
Levy-Valensi, 510
L'Hermite, 156, 227
Lichtenberger, 498
Lindemann, 510
Lorenz, 507
Love, 471

Macpherson, 235
Makrycostas, 495
Malaise, 402
Mallory, 226, 251
Marburg, 156, 430
Marie, 34, 483
Marinesco, 197
Marks, 397
Martin, 430, 538
Masson, 227, 249
Mauss, 194
McAlpine, 407
McKenzie, 547
Menard, 491
Meyer, 507
Mixter, 34, 61, 471, 479
Moore, 164
Müller, 403
Murray, 250
Muskens, 523
Musser, 430
Muthmann, 495

Naffziger, 214
Neri, 432
Newmark, 26
Nonne, 26, 41, 366, 382
Nothnagel, 203

Oberling, 226
Ollier, 519
Ollivier, 201, 202
Oppenheim, 26, 132, 224, 366, 368, 382, 407, 408, 430, 489
Ostertag, 317

Paré, 517
Parrot, 160
Paulian, 197
Pechy, 469
Peers, 233
Pels-Leusden, 314
Penfield, 167, 177, 224, 227, 267, 293
Perman, 495
Petit-Dutaillis, 469
Petrèn, 159, 394
Phillipson, 386
Platt, 264
Polatin, 124
Pool, 514
Potel, 453
Putnam, 543, 550, 551
Puusepp, 511, 551

Queckenstedt, 27

Rademaker, 194
Ransom, 369
Ranzi, 158

Rasmussen, 234, 245, 248, 283, 350, 359
Raven, 264
Ravenel, 211
Raymond, 204, 507, 508
Reichert, 125
Riddoch, 157, 384, 388
Riley, 11
Ritter, 511
Robert, 34
Robinson, 209
Röpke, 453
Ross, 143
Roussy, 226
Rowe, 551
Rowland, 120
Russell, 167, 168, 317, 324

Sachs, 166, 511
Sargent, 221, 503, 504
Sawicki, 210, 436, 451
Schlesinger, 233, 494
Schminke, 112
Schmorl, 469, 471
Schroeder, 215
Schultze, 215, 366, 368, 369, 453
Schwalbe, 168
Selinsky, 195
Serko, 366, 398
Shemkin, 501
Sherrington, 13
Shubert, 324
Sicard, 41, 112
Söderbergh, 408
Solomon, 40
Solovtzoff, 168
Spiller, 226, 316, 388, 430, 511, 538
Steinhard, 365
Steinke, 208, 493
Stertz, 366
Stookey, 27, 469
Stout, 250
Stursberg, 208
Sussman, 55

Tarlov, 248, 277, 284
Taylor, 519
Thomas, 387
Thompson, 369, 394
Tilney, 11, 538
Töpfer, 495
Towne, 125
Turner, 203
Turnesco, 197

Veaudeau, 453
Veraguth, 453, 469
Verocay, 227, 240, 249
Viets, 26
Vincent, 197
Virchow, 263, 469
Vogt, 264

Wakely, 498
Walker, 471, 474
Walsham, 44
Walshe, 143, 384, 389
Weed, 264
Wideroe, 65
Willitt, 44
Wolf, 341
Wolff, 203
Woltman, 200, 201, 204, 217, 343

INDEX OF SUBJECTS

Abdominal distension after laminectomy, 534
Abdominal symptoms in thoracic tumors, 367
Abscess, spinal, roentgen ray diagnosis of, 118
    extradural, 185-189
    intradural, 189
    tuberculous, 119
Air injections, into spine for roentgen localization, 65
Alcohol, intra-arachnoid injection of, for pain, 535-536
Amyl nitrite test for subarachnoid block, 26
Anesthesia, local, for spinal operations, 521, 542
Aneurism, of aorta, 502
  of spinal vessels, 503
  of vertebral artery, 506
Anisocoria, in cervical cord tumors, 407
Anterolateral tracts, division of, for pain, 538
    indications for, 539
    technique, 540-542
Arachnoid, cysts of, 461-467
Arachnoiditis, 193-200
Arnold-Chiari malformation, 50, 167
Arteries, spinal, aneurism of, 503
Arteries and veins, aneurismal varices of, 507
Artery, vertebral, aneurism of, 505-507
Arthritis, of spine, 482
  of vertebrae with spinal tumors, 429
Astroblastoma, pathology of, 300-302
Ataxia, in cord tumors, 386
Athetosis, section of extrapyramidal tracts, 550

Back pain, in giant tumors of cauda equina, 418
  in herniation of nucleus pulposus, 471
  in spinal cord tumors, 370
Bladder disturbances, in complete transverse lesions of cord, 130
  in cord tumors, 388
    nature of, 389
  in tumors of filum terminale, 422, 448
Bone destruction from spinal tumors, 69
Bone proliferation in spinal cord tumors, 75
Brain tumors, spinal metastases from, 316-384, 554
Brown-Séquard syndrome, 402
  in extramedullary tumors, 402
  reversed, 218, 399, 403
    explanation of, 399, 403
Bullet wounds of spinal cord, 153-159

Calcification in tumors, 76
Carcinoma, metastatic of vertebrae, 355, 499
    roentgen ray changes in, 110
Cauda equina, injuries of, 139
  radiculitis of, 204-207
  tumors of. *See* Spinal cord tumors, cauda equina.
Cerebrospinal fluid, alterations in, 38-41
  Froin syndrome, 41
  Nonne syndrome, 41
Cervical neurofibroma, 210, 436
Cervical tumors. *See* Spinal cord tumors, cervical.
Childhood, cord tumors in, 209
Chondroma of vertebrae, 359, 467, 494
  histology of, 360
  roentgen ray diagnosis of, 103

Chordoma, 342, 496
  histology of, 343
  roentgen ray diagnosis of, 106
Chordotomy, for pain, 538
  technique of, 540-542
Cistern puncture (Ayer), 35
Cold, sensation of, in cervical cord tumors, 406
Commissure, myelotomy of, for pain, 543
Constipation in spinal cord tumors, 389
Contractures of muscles, in cord compression, 387
Contralateral tingling, in cervical tumors, 406
Cranial nerve symptoms, in cervical cord tumors, 407
Cutaneous reflexes, centers for, 17
Cutaneous sensibilities, dissociation of, 21, 22
Cysts, echinococcus, 461
  dermoid, 346. *See* Dermoid cysts.
  leptomeningeal, 193
  in nerve sheath tumors, 245
  of spinal cord, 461
    extradural, diagnosis of, 466
      enlargement of spinal canal in, 462
      interpediculate measurements in, 462
      roentgen ray changes in, 99, 100-103
      symptoms of, 465
      operative treatment for, 467

Dermoid cysts, 230, 346
  frequency, 230
  histology of, 346
  roentgen ray changes in, 103
Dentate ligament, anatomy of, 5
  relation of, to spinal cord tumors, 217
Differential diagnosis, of pachymeningitis from spinal cord tumor, 193, 429
  of Pott's disease from cord tumor, 425, 490
  of serous meningitis or arachnoiditis, 430
Dissociation of sensation, in cord tumors, 21, 392
Double spinal puncture, 36-38
Dyspnea, in high cervical tumors, 406
Dystonia, section of extrapyramidal tracts in, 550

Enlargement of spinal canal, with tumors, 73
  in perineurial fibroblastomas, 87-93
Enophthalmos, in cervical cord tumors, 413
Ependymoma. *See* Spinal cord tumors, ependymoma.
Epidermoid, 344
Etiology of spinal cord tumors, 208
Extradural tumor. *See* Spinal cord tumors, extradural.
Extramedullary tumor. *See also* Spinal cord tumors, extramedullary.
  atrophy of muscles in, 380
  differentiation of, from extradural and intramedullary, 435
  dissociation of sensation in, 392
  early motor symptoms in, 382
  removal of, 445
  sensory disturbances in, 390
  tenderness of spinous processes in, 403
Extrapyramidal tracts, section of, for dystonia, 550
Extrusion of intramedullary tumors, 453-455

Fatigability of receptors in cord lesions, 130
Fibrillation of muscles in cord tumors, 380
Filum terminale, tumors of, 422, 448
Fractures of vertebrae, changes in relation of cord and vertebrae in, 126-128
  complete transverse lesion of spinal cord and, 130-136
  due to direct or indirect violence, 124
  hematomyelia and, 137
  hemiparaplegia and, 136
  injury of spinal cord and, 128-135

Fractures of vertebrae—(*Continued*)
  due to metrazol convulsions, 124
  monoplegia and, 137
  partial lesion of spinal cord and, 136-139
  trauma of roots of cauda equina and, 139
Froin syndrome in spinal cord tumors, 41

Ganglioneuroma, histology of, 306-310
Giant tumors of conus and cauda equina, 422
  enlargement of canal in, 95
Girdle sensation in cord tumors, 372
Glioma of spinal cord, 265. *See also* Spinal cord tumors, intramedullary.
  classification of, 267
  intracranial, implantation of, into subarachnoid space, 316-324
  invasion of spinal subarachnoid space by, 314-316
  relative frequency of, 268
Glioblastoma multiforme, histological character of, 294
Gliosis and intramedullary tumors, 325-330, 431
Globulin, increase of, in cerebrospinal fluid, 39
Gumma of spinal cord, 480-482

Hands, atrophy of muscles of, 377
Hemangioblastoma, gross structure of, 229, 332
  histology of, 333-337
Hemangioma of vertebrae, 99, 495
Hematomyelia, 137
Hemilaminectomy, 519
Hemiplegia, spinal, 136
Hemorrhage into spinal cord, 128, 129
Heredity, in cord tumors, 208
Hodgkin's disease, 413, 554
  roentgen ray changes in, 110
Horner's syndrome, 413
Hypernephroma, metastatic, in spine, 427

Incontinence of bladder, in cord tumors, 388, 422, 448
Indication for operations, in spinal cord tumors, 439
Inflammatory diseases of spinal cord, 185-207
Injuries of spinal cord, 123-163
Interpediculate measurements, 71-76, 462
Intervertebral discs, changes in, 75
  in cauda equina tumors, 97
  in ependymomas, 80
  in meningiomas, 84
  in perineurial fibroblastomas, 93
Intervertebral foramina, origin of tumor in, 214
Intramedullary tumors. *See also* Spinal cord tumors, intramedullary.
  appearance of cord in, 445
  Brown-Séquard symptoms in, 402
  comparative frequency of, 209, 210, 222
  differentiation of, from disease of cord, 430, 431
  dissociated sensibility in, 393
  early motor disturbances in, 377
  extrusion of, 453-455
  root hyperesthesias in, 391
Inversion of reflexes, 133, 378
Irremovable tumors, treatment of, 455

Klippel-Feil syndrome, 44-48, 163
Kümmel-Verneuil disease, 55, 484
Kyphosis juvenilis, 53

Lamellar arrangements of fibers, in spinothalamic tracts, 11, 395, 539
Laminectomy, abdominal distension after, 534
  amount of bone removed for ventral growths, 520
  anesthesia in, 521
  bilateral or unilateral, 519
  cervical, headrest for, 522
  decompressive, 455
  extrusion method of, for intramedullary tumors, 453

Laminectomy—(*Continued*)
  hemorrhage during, 529
  historical notes on, 517-519
  locating spines to be removed, 523-524
  osteoplastic methods, 519
  position of patient for, 522
  postoperative treatment, 533-535
  preparation of patient for, 520
  removal of cauda equina growths by, 445-449
    of extradural growths by, 449
    of extramedullary growths by, 445
    of intramedullary growths by, 453-455
  steps of the operation, 524-533
  transdural removal of extradural growths by, 452
  unilateral, 519
  views of Astley Cooper and Charles Bell on dangers of, 517-518
Leiomyosarcoma, pathology of, 354
Leucosarcomatosis, 357
Leukemia, roentgen ray diagnosis of, 110
Level, diagnosis of, in cord tumors, 433-435
Ligamentum flavum, and herniation of nucleus pulposus, 363, 473
  histological changes in, 363-364
  hypertrophy of, 67, 363
Lipiodolography, 61-65
  interpretation of, 114-118
  technique of, 112-114
Lipoma of spinal cord, 230, 349
  gross features of, 230, 350
  histology of, 350, 351
Lumbar puncture, aggravation of symptoms after, 26, 434
  effects of, on cord symptoms, 26
  headache after, 26
  manometric tests in, 27-38
Lymphoepithelioma, roentgen ray changes in, 112
Lymphogranuloma, malignant, 357

Main-en-griffe, in cervical cord tumors, 411, 412
Malignant extradural disease and cord compression, 426
Manometric estimations of spinal fluid pressure, 26-38
Mass reflexes, after cord trauma, 142
  in spinal cord tumors, 384
Medulloblastoma, 304-306
Medulloepithelioma, 312
Meningioma, angioblastic, 263
  calcification in, 83
  effect of, on spinal cord, 127
  frequency of, at various levels, 228
  gross shape of, 225, 226, 254
  gross structure of, 254
  histology of, 255-265
    absence of mitoses, 256
    cells similar to those of arachnoid granulations, 257
    locally invasive, 262
    psammoma bodies, 259
    whorls, 258
  level frequency of, 228
  mechanical effects of, 227
  origin of, 255
  relation of, to spinal cord and dura, 253
  relative frequency of, 228, 252
  roentgen ray evidence of, 84
  size of, 226, 253
Meningitis, circumscribed serous, 430. *See also* Arachnoiditis.
Meningocele, 165
  roentgen appearance of, 51
Metastatic carcinoma, of vertebrae, 499
  lymphosarcoma, 351, 353
  thyroid tumors, 501
Miosis, in cord tumors, 413
Mobility of spinal cord segments, 217
Mortality after operations for cord tumor, 456-458
Motor disturbances, in spinal cord tumors, 372-376
Multiple cord tumors, 554
Myelocele, 165
Myelography, by gas, 65
Myeloid chloroma, 358

Myeloma and cord tumors, 426, 556, 497
  roentgen ray changes in, 112
Myelotomy, for intramedullary tumor, 454
  for syringomyelia, 551
  of commissure, for pain, 543

Neck, pain in, in cervical tumors, 367
Nerve sheath tumors, 234-252. *See also* Spinal cord tumors, nerve sheath; *and* Perineurial fibroblastoma.
Nerves, cranial, affections of, in cervical tumors, 407
Neuroblastoma, 311
Neuroepithelioma, 310
Neurofibroma, cervical, 210, 215, 436
  histological features of, 248, 249
  multiple, in von Recklinghausen's disease, 248
    roentgen ray changes in, 106
Neurofibromatosis, 248
Nonne syndrome, in cord tumors, 41
Nucleus pulposus, herniation of, 361-364, 467-480
    backache in, 471
    etiology of, 361
    frequency of, 471
    gross features of, 362, 472
    histological features of, 362
    hypertrophy of ligamentum flavum and, 363, 473
    lipiodol, for diagnosis of, 475
    lumbar puncture in, 474
    operations for, 476-479
      results, 479
    situation of, 472
    symptoms in, 474
    x-ray in, 57-61, 474
Nystagmus, in high cervical tumors, 407

Oculo-pupillary symptoms in low cervical tumors, 413
Oligodendroglioma, 297-300
Operation, indications for, 439. *See also* Laminectomy.
  for fractures of spine, 149-153
Operation—(*Continued*)
  for relief of pain, 535-543
  for spinal cord tumors, 524-543
Osteoma of vertebrae, 494

Pachymeningitis, 189-193
Paget's disease, 120, 484
  roentgen ray diagnosis in, 120
Pain, absence of, in cord tumors, 365
  as an early symptom of cord tumor, 367
  in back, in giant tumors, 418
    in spinal cord tumors, 19, 370
    in tumors of conus and cauda equina, 418
  division of anterolateral column for, 538
    of posterior spinal roots for, 536
  fibers which transmit, 538
  intra-arachnoid injections of alcohol for, 535
  operations for relief of, 535-543
Paresthesias, in cord tumors, 370. *See also* Spinal cord tumors, paresthesias in.
Pathology of cord tumors, 231-365
Pedicles, roentgen ray changes in, 80
Perineurial fibroblastoma. *See also* Spinal cord tumors, Perineurial fibroblastoma, Nerve sheath tumors.
  enlargement of spinal canal in, 87-93
  gross structures of, 226, 228
  histology of, 237-252
  mechanical effects of, 227
  palisades in, 240
  pathology of, 235, 252
  roentgen ray diagnosis in, 87-93
Phrenic nerve, involvement of, in high cervical lesions, 132, 407
Pilomotor reflexes, in cord compression, 388
  in complete transverse lesions of cord, 130
Polar spongioblastoma, 302-304
Posterior root section, for pain, 536
  for spasticity, 544-546
  for torticollis, 546-549
Pott's disease, 425, 485-493

Radiculitis, of cauda equina, 204-207
Radium therapy, in tumors, 456
Recklinghausen's disease, 554. *See also* Neurofibromatosis.
  roentgen ray changes in, 106
Reflexes, inversion of, 378
Réflexes de défense, and lower end of cord tumor, 385
Reversed Brown-Séquard syndrome, 399, 403. *See also* Spinal cord tumors, reversed Brown-Séquard, and Brown-Séquard.
Rhizotomy, for pain, 536
  for spasticity, 544
  for torticollis, 546
Roentgen ray diagnosis, 42-122
  of gas myelography and, 65
  of Arnold-Chiari malformation, 50
  of bone destruction, in tumors, 69
  of bone proliferation, 75
  of calcification, in tumors, 76
  of calcium deposits in meningiomas, 83
  of congenital anomalies, 44-49
  of cysts, 99, 100-103
  of enlargement of spinal canal, in ependymomas, 80
    in giant tumors of cauda equina, 95
    in perineurial fibroblastomas, 87-93
  of extradural tumors, 98
  of hemangioma, 99
  of intervertebral discs, changes in, 75
  of Klippel-Feil syndrome, 44
  of Kümmel's disease, 55
  of Kyphosis juvenilis, 53
  of ligamentum flavum, 67
  of lipiodolography and, 61-65
  of lipomas, 76
  of meningiomas, calcium deposits in, 83
  of meningocele, 51
  of nucleus pulposus, herniation of, 57-61
  of pedicles, 70
    interpediculate measurements, 71
Roentgen ray diagnosis—(*Continued*)
  of perineurial fibroblastoma, 87-93
  roentgen ray appearance, of chordoma, 106
    of cysts, idiopathic, 100-103
    of giant cell sarcoma, 109
    of Hodgkin's disease, 110
    of leukemia, 110
    of lipoma, 104
    of metastatic carcinoma, 110
    of myeloma, 112
    of neurofibromatosis, 106
    of osteogenic sarcoma, 111
    of osteoma, 112
    of Paget's disease, 120
    of Schminke tumor, 112
    of spinal abscess, 118
    of tuberculous abscess, 119
    of xanthomatosis, 120
  of spondylolisthesis, 49
  of vertebral column fractures, 51
  of vertebral column dislocations, 57
Root anesthesias in tumors of cauda equina, 420
  character of pain with, 365-370
Root pain, frequency of, in cord tumors, 365-370
  increase of, after lumbar puncture, 26
  in lumbar and sacral tumors, 420
  in Pott's disease, 487
  in spinal tumors, 365-370

Sarcoma, giant cell, of vertebrae, 497
  multiple, of spinal cord, 556
    roentgen ray changes in, 94
  reticulum cell, 354
Schminke tumor, 112
Segment localization of muscles, 14-17
Sensory disturbances, in tumors of conus and cauda equina, 419
Sensory levels in spinal diseases, 22
Sensory progression, in extramedullary tumors, 390
Shoulder, pain in, in cervical tumors, 367
Spasticity, rhizotomy for, 544-546

Spina bifida, 163-184
  Arnold-Chiari malformation in, 167
  associated anomalies, 167-170
  classification of, 164
  frequency of, 164, 166
  meningocele, 165
  myelocele, 165
  operative results in, 179
  operative treatment for, 173-179
  symptomatology of, 171-173
Spina bifida occulta, 180-184
  hypertrichosis in, 181
  lipoma in, 181
  symptomatology of, 182, 183
  treatment for, 183, 184
Spinal accessory nerve, division of, for torticollis, 546
Spinal cord, abscess of, 118, 200-204
  anatomy of, 1-17
  anterolateral tracts of, 10
  arteries of, aneurism of, 503
  blood vessels of, aneurismal varices of, 507
  calcification in, 76
  centers in, for skin and tendon reflexes, 17
  complete transverse lesions of, 120-135
    at 4th to 6th cervical segments, 132
    at 7th cervical segment, 133
    at lumbar and sacral levels, 135
    at thoracic levels, 134
    diagnosis of, 140-142
    indications for operation in, 150-152
    prognosis in, 145, 146
    treatment for, 147-153
  fiber tracts in, 9
  fibers in, for bladder and rectum, 13
  gumma of, 480
  hematomyelia of, 137
  inflammatory disease of, 185-207
  injuries of, 123-163
    from bullet wounds, 153-159
    electrical, 162
    from fracture of spine, 125
Spinal Cord—(*Continued*)
  injuries of—(*Continued*)
    obstetrical, 161
    operative treatment, 152
    partial, 136-138
    stab wounds, 156-160
  lamellation of spinothalamic tracts, 11
  relation of segments of, to vertebrae, 6
  veins of, varicosities of, 509
Spinal cord tumors, aneurism and, 503-507
  ataxia in, 386
  bladder disturbances in, 388
  Brown-Séquard syndrome in, 402
  cauda equina, 418-422
  cervical, atrophy of intrinsic muscles of hands in, 377
    rigidity of neck in, 367
  cervical I to III, 406-408
    atrophy of muscles of shoulder girdle in, 408
    atrophy of trapezius and sternomastoid in, 407
    character of pain in, 406
    difficulty of breathing in, 406
    facial muscles, weakness of, in, 407
    mode of onset of, 406
    numbness of cheek in, 407
    nystagmus in, 407
    papilledema in, 408
    sense of coldness in lower limb in, 406
    sensory disturbances in, 408
  cervical IV to VI, 408-412
    atrophy of hand muscles in, 410
    main-en-griffe in, 411
    pain in shoulder in, 367, 408
    sensory disturbances in, 411
    spinal hemiplegia in, 409
    symptoms, early, 408
  cervical VII to thoracic I, 412-414
    atrophy of hand muscles in, 412
    dissociation of sensations in, 414
    Horner's syndrome in, 413
    oculo-pupillary symptoms in, 413

Spinal cord tumors—(*Continued*)
  in childhood, 209
  classification of, 232
  contractures in, 387
  course of, atypical, 376
    typical, 375
  cysts. *See* Cysts.
  dentate ligament, relation of, to, 217
  diagnosis, 415-438
    from arachnoiditis, 430
    from benign bone tumors, 429
    from hypernephroma, 427
    of level of, 433-435
    from malignant disease of vertebrae, 426
    from metastatic carcinoma, 427
    from multiple myeloma, 428
    of multiple tumors, 554-558
      regional, 432-438
    from Pott's disease, 425
    from syringomyelia, 431
  double spinal puncture in, 36-38
  dumb-bell shaped, 214, 215
  effects of, on nerve roots, 224
  enlargement of spinal canal in, 73, 80-93
  ependymoma, 268
    enlargement of canal in, 80
    gross features of, 270-272
    histological character of, 272-280
      blepharoplasten in, 274
      occurrence of, at different levels, 268-269
      rosettes, 279
      theories of origin of, 282
  etiology of, 208
  extradural, 213
    and intradural, 452
    dumb-bell shaped growths, 214, 215
    frequency of, 210
    primary, 213
    removal of, 449
    roentgen ray changes in, 98-100
    secondary, 213
    transdural removal of, 452

Spinal cord tumors—(*Continued*)
  extramedullary. *See also* Extramedullary tumors.
    dissociation of sensations in, 392
    early motor disturbances in, 382
    muscle atrophies in, 378
    peripheral dermatomes most affected, 394
    progression of sensory disturbances in, 390
    technique of removal of, 445
      of ventral growths, 444
    tenderness of spinous processes in, 403
    types of sensory disturbance in, 396, 398
  fibrillation of muscles in, 380
  frequency of, 209, 210, 232
    and brain tumors, 233, 234
    and length of cord, 211
  giant tumors, 422
  girdle sensation in, 372
  hemangioma, 495
  heredity in, 208
  interpediculate measurements, 71, 423
  intramedullary, appearance of cord in, 445
    Brown-Séquard syndrome in, 402
    differentiation of, from disease of cord, 430
    early motor disturbances in, 377
    frequency of, 22, 207, 210
    root hyperesthesias in, 391
    technique of removal of, 453-455
  involvement of limbs in, order of, 381
  irremovable, 455
    mass reflexes in, 384
    motor disturbances in, appearance of, 372, 376
    order of involvement of limbs in, 373
  nerve sheath, 234-252. *See also* Perineurial fibroblastomas.
    connective tissue in, 247
    edema between cells in, 244
    frequency of, 234

Spinal cord tumors—(*Continued*)
  nerve sheath—(*Continued*)
    histology of, 237-252
    neurofibroma, 248
    nuclei of cells in, 243
    occurrence of cysts in, 245
    onion bulb appearance of, 239
    origin of, 249
      theories of, 249-252
    palisades, 240
    relation of, to dura, 235
    size of, 228, 235
    vascularity of, 246
  numbness, subjective, in, 372
  operations for, 439-456
    division of anterolateral tracts for pain in irremovable growths, 538
    exposure of, 440
    exposure of ventral growths, 444
    indications for, 439
    laminectomy, decompressive, 455
    mortality after, 456-458
    prognosis in, 458
  order of involvement of limbs in, 373
  osteoma, 494
  pain in back in, 19, 370
  paresthesias in, 370
  and pregnancy, 208
  reflex sweating in, 388
  regional diagnosis in, 432
  relation of, to cord and membranes, 212, 216
  removal of, technique of, 445-449
  reversed Brown-Séquard syndrome in, 399, 403
  roentgen rays in, 68-122
  root pain in, 367-369
  sensory disturbances in, 390-402, 419
  size of, 209
  spasticity in, 381
  spinous processes, tenderness of, 403
  symptoms at onset, 365, 366
  thoracic. *See also* Thoracic tumors.
    indefinite sensory levels in, 396
    level signs of, 396
    root pain in, 367

Spinal cord tumors—(*Continued*)
  thoracic II to VIII, 474
    abdominal reflexes in, 415
    intercostal neuralgia in, 367
    pain simulating gall bladder disturbance in, 367
    pain under shoulder blade in, 367
    vague upper level frequent, 415, 416
  thoracic VIII to XII, 416
    abdominal reflexes in, 416, 417
    xanthochromia in, 38, 417
  treatment of, 439-461
  trophic ulcers in, 420
  types of history, 375, 376
  vascularity of, 448
  ventral growths, 397, 438
  xanthochromia in, 38
Spinal hemiplegia, 136
Spinal membranes, anatomy of, 5
Spinal nerve roots, anatomy of, 2, 3
  injuries of, 123-163
Spinal puncture, double, 36-38
Spinothalamic tracts, lamellation of, 11, 396, 539
Spinous processes, tenderness of, 403
Spondylitis, tubercular, 485, 493
Spondylolithesis, 49
Spondylose rhizomélique, 483
Subarachnoid block, double spinal puncture in, 34-38
  manometric tests in, 26-38
  puncture of cisterna magna in, 35
Subarachnoid space, invasion of, by gliomas, 314-316
  implantation of tumors in, 316-324
Syphilis and spinal cord tumors, 425, 480
Syringomyelia, myelotomy for, 551

Tendon reflexes, centers for, 17
Teratoma, 348
Thoracic tumors. *See* Spinal cord tumors, thoracic.
  vague levels in disease of, 23, 415
Torticollis, rhizotomy for, 546
Transverse lesions of spinal cord, 130-135

Traumatic hysteria after injuries of spinal cord, 24
Triceps reflex, inversion of, 378, 412
Trigeminus, rarely involved in cervical cord tumors, 407
Trophic disturbances, in cord lesions, 147-153
  in complete transverse lesions, 130
Tuberculosis of vertebrae, 485-493
  symptoms and diagnosis, 487-489
  treatment of, 491-493

Varicosities of spinal veins, 509-516
  diagnosis of, 513
  roentgen ray changes in, 94
  symptomatology of, 512
  treatment for, 514-516
Ventral tumors, 397, 438
  technique of removal, 444
Vertebrae, arthritis of, 482
  chondroma of, 467, 494
  chordoma of, 496, 554
  giant cell sarcoma of, 497
Vertebrae—(*Continued*)
  hemangioma of, 77, 495
    roentgen ray changes in, 77
  metastatic tumors of, 499-502
  myeloma of, 497-499, 554
  osteoma of, 494
  primary tumors of, 493
Vertebral column, anatomy of, 1, 123
  dislocations of, 125, 127 et seg.
  mechanics of, 123
    roentgen ray changes in, 57
  fractures of, due to convulsions, 124
    etiology of, 124
    roentgen ray changes in, 51-53
Vertebral levels of spinal cord tumors, 211

Xanthochromia, 38
  frequency of, in spinal cord tumors, 39
Xanthomatosis, roentgen ray changes in, 120

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